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When a "Fourth Meal" Appears Advantageous

There are many instances in pediatric practice when the patient's nutritional state must be improved, but when it is difficult to prompt an increased food intake at the regular meals. In such instances cereal foods (prepared or to be cooked), between meals or at bedtime, present an excellent means of bringing total daily food consumption to the desired higher level.

The appended table indicates what percentages of the daily requirement of essential nutrients are provided by such a "fourth meal," consisting of 1 ounce of cereal (whole-grain, enriched, or restored to whole-grain values of thiamine, niacin, and iron), 4 ounces of whole milk, and 1 teaspoonful of sugar. The values shown below for cereals and milk are composite averages.

	Total Daily Allowances of Child 4-6*	Supplied by Dish of Cereal, Milk, and Sugar	Percentage of Total Daily Allowances
Calories	1600	205	12.8%
Protein	50.0 Gm.	7.0 Gm.	14.0%
Calcium	1.0 Gm.	0.16 Gm.	16.2%
Iron	8.0 mg.	1.29 mg.	16.2%
Vitamin A	2500 I.U.	187 I.U.	7.5%
Thiamine	0.8 mg.	0.18 mg.	22.1%
Riboflavin	1.2 mg.	0.30 mg.	25.1%
Niacin	8.0 mg.	0.64 mg.	10.5%

* Based on "Recommended Daily Allowances" of the National Research Council for a child 4 to 6 years of age.

The presence of this seal indicates that all nutritional statements in this advertisement have been found acceptable by the Council on Foods and Nutrition of the American Medical Association.

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The Journal of Pediatrics

VOL. 24 MAY, 1944 NO. 5

Original Communications

THE PROPHYLACTIC USE OF SULFANILAMIDE IN CHILDREN WITH INACTIVE RHEUMATIC FEVER

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NEW YORK, N. Y.

IN THE fall of 1939, following reports[1, 2] of studies of the prophylactic use of sulfanilamide in patients with inactive rheumatic fever, a similar investigation was undertaken in the Children's and Adolescent Cardiac Clinic of Bellevue Hospital.

Thomas and France[1] and Coburn and Moore[2] reported the prevention of Group A hemolytic streptococcus infection and rheumatic relapses with the administration of small daily doses of sulfanilamide throughout the winter months to susceptible rheumatic individuals. Although the results of these authors were striking, no comparable control group was studied by Coburn and Moore, and the patients of Thomas and France comprised a relatively less susceptible age group.

It was therefore thought important to study the value of prophylactic sulfanilamide in a younger and presumably more susceptible age group and to determine its safety and the feasibility of its administration to a large group of ambulatory clinic patients from the lowest economic background. A study at Bellevue Hospital might be expected to throw additional light on both these phases of the problem, since the clinic is a large and active one, drawing from a district in which rheumatic fever is prevalent. With long-term convalescent facilities as limited as they are, rheumatic subjects living under similar conditions of everyday exposure in crowded urban areas represent all but a small proportion of rheumatic patients in the country. Any prophylactic agent to be really effective against this disease must be practical to administer and effective under these conditions.

From the Children's Medical Service, Bellevue Hospital, and the Department of Pediatrics, New York University.

This study was supported by a grant from The John and Mary Markle Foundation, New York, N. Y.

Sulfanilamide used throughout the study was supplied by the Winthrop Chemical Co., Inc., New York, N. Y.

PLAN OF STUDY

The study covered a period of four years. During the first season (1939-1940) fifteen children with advanced heart disease received the drug in small daily doses for an average of seven months and were watched closely for signs and symptoms of respiratory infection, rheumatic relapses, and toxicity. No special control group was followed during this season. The study was continued and enlarged during the following three years, and the number of children taking the drug was increased to bring the numbers of individuals and patient-seasons to a size of statistical value. A group of rheumatic children with comparable socioeconomic backgrounds studied by Dr. Homer Swift and his associates at the Hospital of the Rockefeller Institute served as a control series during the second and third years. During the fourth season the control group consisted of patients from Bellevue Clinic who had previously taken the drug. Both groups were followed closely by both clinical and laboratory means for evidence of infections with Group A hemolytic streptococcus and rheumatic relapses.

Sulfanilamide was employed throughout the study. In 1939 it was the most thoroughly studied sulfonamide. It was subsequently preferred to the other sulfonamide drugs because it was used by other investigators in this field, thus making it possible to pool and compare our results with theirs.

Number of Cases.—Eighty-eight patients (fifty-one girls and thirty-seven boys), ranging in age from 6 to 18 years, were given the drug during the fall, winter, and spring months for a total of 181 patient-seasons. Seven children received the drug for four seasons, nineteen for three seasons, forty-eight for two seasons, and thirty for one season. Nine of the children taking the drug for more than one year received it for one season at Irvington House, and one child received it for two seasons there; these patients have also been included in the report by Kuttner and Reyersbach.[3] Nine additional children were started on the drug but were dropped from the study because they were uncooperative or because adequate supervision was not possible.

Selection of Cases.—

The Treated Group: Fifty-eight (66 per cent) of the eighty-eight children were between 11 and 15 years of age at the time of first administration of the drug. Sixty-four per cent were under 14 years. The majority of cases represented severe rheumatic disease. Fifty-three per cent of the children had had three or more attacks of rheumatic fever and only 25 per cent had had only a single attack. In 93 per cent the most recent attack had occurred within three years, and in no instance had it occurred more than five years previously. Cardiac diagnoses ranged from potential heart disease to the severest type of stenosis and insufficiency of the mitral and aortic valves. Sixty-six per cent had mitral diastolic murmurs or combined mitral and aortic disease.

Early in the study children with histories of very frequent attacks and with signs of advanced heart disease were selected. Necessarily such a group contained many older children and adolescents. Later, a group of younger children with a history of only one recent attack was added. These were all within the most susceptible age group. It has been our impression, however, that those children who have had many attacks and have already developed signs of severe cardiac damage are more likely to have relapses, even though they are beyond the most susceptible age, than are young children with a history of only one attack and without evidence of cardiac damage.

The Control Group: During the second and third seasons the control group consisted of one patient followed in the Bellevue Clinic and sixty-three patients observed for 100 patient-seasons at the Hospital of the Rockefeller Institute. These children were of approximately the same socioeconomic background as those of the Bellevue group. (About 25

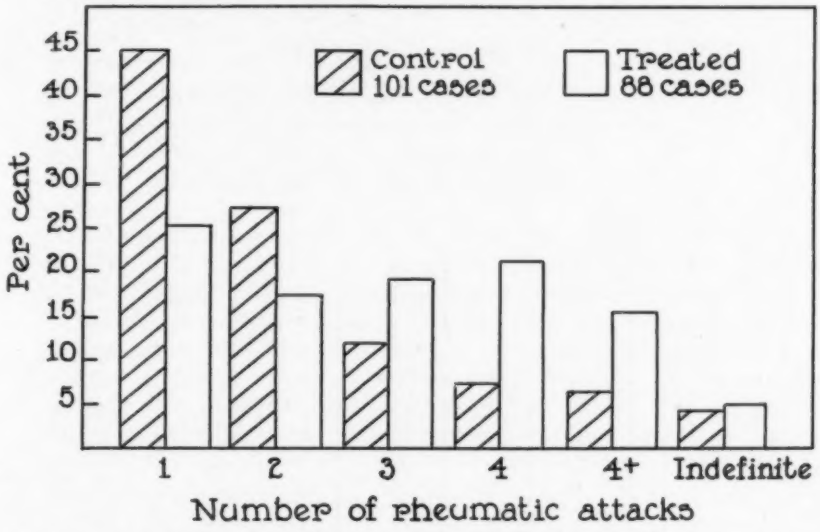

Fig. 1.—Comparison of the number of rheumatic attacks in children in the control and treated groups before period of study.

per cent of them had been "loaned" by the Bellevue Clinic to the Rockefeller Institute Hospital for purposes of their study.) However, they were slightly younger (93 per cent were under 14 years at the time of first observation as a control) and had somewhat less severe rheumatic fever; more than one-half of them had had one attack only, and only one-half of them had evidence of organic heart disease. During the fourth season the group consisted of thirty-seven children who had taken the drug during one or more previous seasons. The children in this group were more nearly comparable to those in the treated group in regard to age and severity of the rheumatic disease. (Figs. 1 to 3 illustrate the

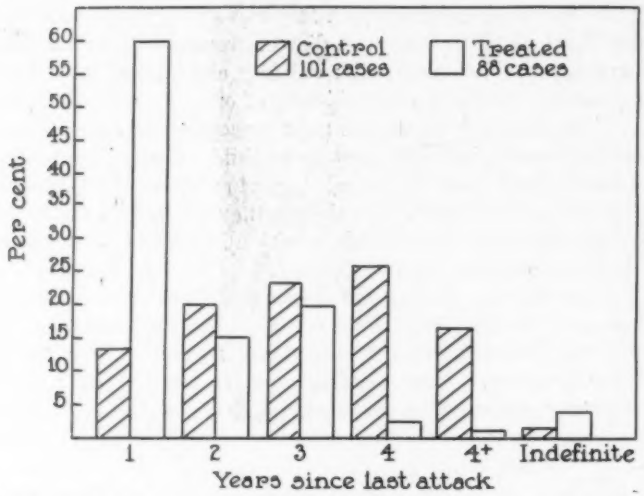

Fig. 2.—Comparison of time interval in years between last rheumatic attack and beginning of study of children in control and treated groups.

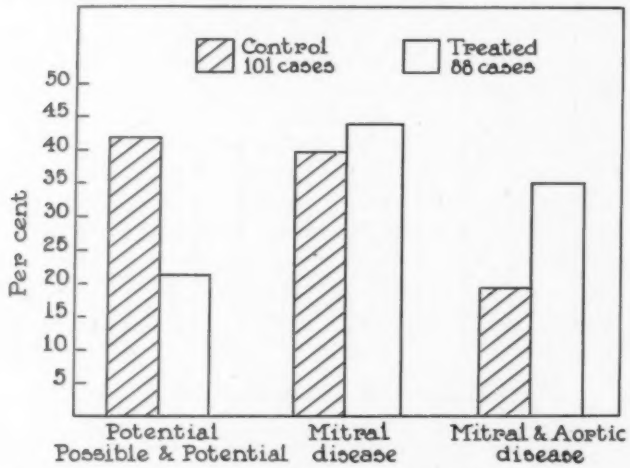

Fig. 3.—Incidence of cardiac involvement in children in control and treated groups.*

difference between those in the sulfanilamide and those in the control groups and show that the children given the drug had more severe rheumatic disease.)

Clinic Procedure.—

The patients were followed by the same two physicians throughout the first three years, and by one of these two through the fourth season.

Before starting the drug each patient received a thorough physical examination. The following laboratory data were obtained: a complete blood count,

*Diagnoses were made according to the criteria for the Classification and Diagnosis of Heart Disease: Heart Committee of the New York Tuberculosis and Health Association.

erythrocyte sedimentation rate, blood antistreptolysin titer, nose and throat cultures, and urinalysis. If no evidence of rheumatic activity was found, the treatment was outlined to the mother and child, with emphasis on the necessity of regularity in taking the drug and of reporting to the clinic on schedule. A special appointment card was given with written instructions to report at the clinic or to telephone the hospital should fever, rash, nausea, vomiting, malaise, sore throat or cold, nosebleeds, or joint pains develop.

The children were seen twice a week for the first three weeks, once a week for the following two weeks, and biweekly thereafter. Whenever indicated because of symptoms or suspicious laboratory findings more frequent clinic visits were made. A certain number of children were seen in their own homes when too ill to come to the clinic or when contagious disease was suspected. After administration of the drug was discontinued in the summer, each child was seen twice during the following month.

At each clinic visit weight, temperature, and pulse were checked, and the child was examined for signs of drug toxicity, upper respiratory infection, and rheumatic activity. Hemoglobin and white blood cell counts were taken at every visit. Differential counts were made at every visit until the last season, when they were done less often. Erythrocyte counts and sedimentation rates and antistreptolysin titers were determined once a month. During the second year nose and throat cultures were taken monthly or in the presence of signs of infection. During the last two years they were taken on every routine clinic visit. Hemolytic streptococci recovered were typed by Dr. Robert F. Watson of the Rockefeller Institute Hospital.

Dosage of Drug.—Our aim was to maintain a concentration of the drug in the blood of from 1.5 to 3 mg. per cent. In general it was found that 1.3 Gm. (20 gr.) daily for those under 110 pounds and 1.6 Gm. (25 gr.) daily for those over 110 pounds was necessary. Three of the smallest children received 1 Gm., and during the first two seasons seven of the largest adolescents required 2 Gm. (30 gr.) a day. The daily amount was divided into three doses, taken before breakfast, at 12 or 3 P.M., according to the school schedule, and at bedtime. All except the three smallest children were started on a daily dose of 1 Gm., and this was increased after from three to seven days to 1.3 Gm. A final increase to 1.6 Gm. was made after another week for those in whom the blood concentration was low.

TOXIC MANIFESTATIONS

Symptoms of drug toxicity were insignificant. Two children had slight itching rashes for one or two days which cleared while they remained on the drug. In two others faint cyanosis was observed from time to time throughout the period of chemoprophylaxis. Three children reported slight nausea once or twice during the first week on the drug. Four reported the following subjective complaints: "dazed" the first day; "drowsy" and "dizzy" the first few days; and "tired" during the first few weeks. There were no febrile reactions and no vomiting.

Effect on General Health.—The prolonged administration of sulfanilamide had no apparent effect on the weight and general health of the children. In none was the appetite affected over any length of time.

Ten children failed to gain at the expected rate. The majority, however, gained normally. The common cold appeared with about the expected frequency. In a few instances sore throats accompanying colds were quite severe, without, however, hemolytic streptococci being cultured from the throats. A variety of other infections occurred while the children were taking the drug regularly. There were eight children with German measles, one each with varicella, pertussis, and herpes zoster. Severe tracheitis and bronchitis requiring hospitalization occurred in two children, and there were two with otitis media. One child developed lobar pneumonia, and another had acute appendicitis and an appendectomy. Four children had teeth extracted uneventfully.

Effect on the Blood Picture.—No changes took place in the blood picture sufficient to indicate discontinuance of the drug permanently, although in several patients it was temporarily discontinued, because of a fall in total white or granulocyte count. On readministering the drug to these children this fall did not recur.

Hemoglobin and Red Blood Cell Count.—Hemolytic anemia did not occur. In less than 25 per cent of the children there was a slight drop in hemoglobin, accompanied in a few instances by a corresponding drop in red blood cell count, during the first six to eight weeks on the drug, followed by a gradual rise during the subsequent months.

White Blood Cell Count and Differential.—Acute agranulocytosis did not occur, and in none of the children did the total or granulocyte count fall dangerously low. During the four seasons white blood cell counts below 5,000 occurred in forty-one children; roughly, one-third of these were encountered during the first two months of treatment. Granulocyte counts between 40 and 50 per cent were even more frequent and occurred at any time during the season. In twenty-three cases granulocytes of less than 40 per cent were found, and in fourteen children the count was less than 35 per cent. The lowest was in a 9-year-old boy, who, during his sixth week of treatment, showed 12 per cent granulocytes in a total count of 4,100. The drug was immediately discontinued and the count followed daily. During the next ten days it gradually rose to normal, with a high proportion of young forms. Sulfanilamide was then readministered at one-half the previous dose and was well tolerated. During his subsequent three seasons on the drug the boy remained well with no recurrence of neutropenia.

In a few other instances in which the total white blood cells, as well as the granulocytes, were low, the drug was temporarily discontinued until the granulocyte count had risen. The majority of these children with relatively low granulocyte counts continued, however, to take the drug while kept under close observation; a white blood count in some patients was done as often as twice a day. In no instance did the leucopenia increase, even under continued administration of the drug, although a number of children tended to run granulocyte counts between 35 and 45 per cent throughout a greater part of the season.

Our impression from this study of the leucocyte counts is that healthy children can, and probably quite frequently do, run low total counts with a relatively low proportion of granulocytes during the winter months. We believe that unnecessary alarm has been caused by total counts of from 4,000 to 5,000 with granulocytes of from 35 to 50 per cent. We do not mean to imply that the rare occurrence of a real agranulocytosis should not be watched for or that the total white blood cell count should not be carefully followed in children taking any of the sulfonamide drugs. The danger of a rare case of sensitivity is always present. One such case has been reported by Stowell and Button[4] in a 12-year-old boy, who, in his fifth week of treatment, developed a sore throat, fever, and a rapidly fatal acute agranulocytosis. Of interest in this case is the fact that the boy had been having blood counts regularly twice a week and had had a normal total white and differential count three days before the onset of fever and sore throat and six days before his admission to the hospital, when he was found to have a white count of 300 with 1 per cent polymorphonuclear cells.

Effect on the Urine.—Routine urinalysis revealed no evidence of renal irritation. In four patients albuminuria present before taking the drug remained unchanged during the period of sulfanilamide prophylaxis. In two of these patients nephritis had been diagnosed previously.

Effect of Prolonged Administration of Sulfanilamide on Subsequent Use of Other Sulfonamide Drugs.—There has been some question whether the prolonged use of small doses of sulfanilamide might not modify the reaction of an individual to the subsequent administration of other sulfonamides for therapeutic purposes. We have observed prompt and dramatic response to sulfadiazine in two patients with pneumonia. One was a boy who developed a type I pneumococcus lobar pneumonia during a brief period when he was not taking sulfanilamide. The other was a girl who, while taking the drug, developed a severe respiratory infection, fever, and physical and x-ray findings suggestive of consolidation. Type VIII pneumococci were cultured from her throat.

Effect of Temporarily Interrupting Sulfanilamide Prophylaxis.—The possibility of developing sensitivity to sulfonamides has been emphasized in a number of recent publications. Lyons and Balberor[5] report severe febrile reactions which they ascribe to hypersensitivity to the drug in 36 per cent of patients receiving a second course of sulfathiazole. Our evidence on this subject is interesting. In several patients in the treated group lapses of from one to ten days occurred when the drug was not taken. Among those who were finally dropped from the study because of lack of cooperation were four children who were restarted on the drug from one to three times at intervals up to three months. Fifty-eight children began taking the drug for a second, third, or fourth time after the summer interval of four or five months. In none of these instances of giving the drug for the second to fourth time after intervals of from a few days to several months was there any evidence of sensitization or increased toxicity of the drug.

RESULTS

The Incidence of Hemolytic Streptococcal Infections and Rheumatic Relapses.—

Control Group: During the four years of the study, in the control group of 101 children followed for 135 patient-seasons, there were fifty-four infections with Group A hemolytic streptococcus. In addition there were fourteen children who had nose or throat cultures positive for the streptococcus but who showed no evidence of infection. There were twenty-six rheumatic relapses (nineteen definite with two deaths, and seven possible), an incidence of 18.8 per cent; three children showed continuing signs of rheumatic activity throughout the period of the study, and one child died of subacute bacterial endocarditis.

In addition there were nineteen children who had taken the drug for one or more seasons, who were dropped from the study and not followed as controls. Four of these disappeared from observation. Of the remaining fifteen, six suffered relapses during subsequent years within the period of the study.

Treated Group.—In the group of eighty-eight children given sulfanilamide for a total of 181 patient-seasons there were five infections with Group A hemolytic streptococcus. In three of these patients the diagnosis of infection was based on a marked rise in antistreptolysin titer only. In two of these three definite rheumatic reactivation occurred.

J. R., a 13-year-old boy with long-standing and advanced heart disease, had run a perfectly smooth course on the drug from December, 1939, to mid-June, 1940, maintaining a good blood level of the drug throughout. Without antecedent respiratory or other demonstrable infection, he developed pain in the groins, fever, and, two days later, smoky urine. On admission to the hospital his heart was overactive and irregular (electrocardiogram showed a prolonged P-R interval and dropped beats), the erythrocyte sedimentation rate was markedly elevated, and the urine was loaded with red blood cells, casts, and albumin. The signs of nephritis subsided moderately, but rheumatic activity progressed through pericarditis and failure, to death six months after admission. Autopsy findings confirmed the diagnosis of nephritis and rheumatic activity.

This case occurred during the first season of the administration of sulfanilamide, when nose and throat cultures and antistreptolysin titers were not being routinely studied. However, a high antistreptolysin titer was found after admission to the hospital.

A. B. was a 15-year-old girl with a long history of rheumatic fever and advanced heart disease, whose cardiac reserve was sufficiently diminished to require digitalis. During her second season on the drug she did well for six months, although she had three mild colds. The middle of May her erythrocyte sedimentation rate and antistreptolysin titer were normal and she had no complaints. She was seen again two weeks later, without complaints and with a normal leucocyte count. On her next visit to the clinic, June 9, her erythrocyte sedimentation rate was markedly elevated, antistreptolysin titer had risen, and she had a leucocytosis of 13,500, with 84 per cent polymorphonuclear leucocytes. Hemolytic streptococci were not cultured from her nose or throat, and she was still without symptoms. Six hours later there was a sudden onset of symptoms of fever and malaise, followed by dull precordial pain and pain in the knees. The color be-

came poor and the liver tender and more enlarged than previously. These symptoms gradually subsided over a period of ten days, although the sedimentation rate and antistreptolysin titer were still elevated the end of July when the special clinic closed until fall.

There is no doubt that these two cases represent failure of the drug to protect against hemolytic streptococcus infection and rheumatic relapses. Even though no previous antistreptolysin titer had been determined in the first case, it is reasonable to assume that the very high titer obtained after the boy's admission to the hospital with acute hemorrhagic nephritis and signs of active carditis, indicated streptococcal infection while the boy was taking sulfanilamide. Although the second patient had no recognized infection with a hemolytic streptococcus, she must have had a so-called silent infection between May 13 and June 19. It is probable that repeated nose and throat cultures during this period would have revealed the presence of this organism.

Besides the two definite cases of reactivation just discussed, there were two other children with very advanced heart disease who died of congestive failure without clinical or laboratory evidence of either hemolytic streptococcal infection or of rheumatic activity. At postmortem examination there was microscopic evidence of active rheumatic myocarditis in both instances.

M. G., a 15-year-old boy, had a much enlarged heart and mitral and aortic stenosis and insufficiency. Early in March of his second season on the drug he was seen in the clinic and found free of clinical and laboratory evidence of rheumatic activity. One week later he was admitted to the hospital with severe congestive failure. He died two days after admission without developing further signs of infection. His antistreptolysin titer was normal in the hospital as it had been throughout his clinic observation.

D. S., a 13-year-old girl whose rheumatic history dated from scarlet fever at the age of 4 years, had had congestive failure at 8 years of age and had been taking sulfanilamide prophylactically since the age of 10 years. Between her third and fourth seasons she again had congestive failure and developed auricular fibrillation. She was digitalized and again started on the drug. However, her condition did not improve; she grew gradually weaker and in the middle of the winter her condition grew rapidly worse and she died. During this period she showed no evidence of rheumatic activity and her antistreptolysin titer remained low.

In addition to the four cases discussed there were two children who developed signs of rheumatic activity within two weeks of starting the drug in the fall and four children who had transient rises in their erythrocyte sedimentation rates without definite signs of rheumatic activity within a short time of starting the drug in the fall. The two children who relapsed within two weeks had both had signs of active infection a few weeks before starting the drug and undoubtedly represent cases of continuing rheumatic activity rather than reactivation of a quiescent infection. In view, however, of the latent period between

streptococcal infections and rheumatic reactivation, such an occurrence during the first two weeks of the administration of sulfanilamide could hardly be considered as evidence of failure of the drug to protect.

Thus there were two definite and two possible rheumatic relapses in 181 patient-seasons of the prophylactic administration of sulfanilamide, an incidence of 2.2 per cent, compared to twenty-six relapses (nineteen definite and seven possible), or 18.8 per cent in the 138 patient-seasons of the control group (Fig. 4). Of all the patients in our study, the two in the treated group with definite relapses represent, in our opinion, the only ones in whom there was real failure of the drug to protect. In the other two children cardiac failure on a mechanical basis was probably the chief cause of death, although it is quite possible that rheumatic individuals of this type are never really free from low-grade active disease.

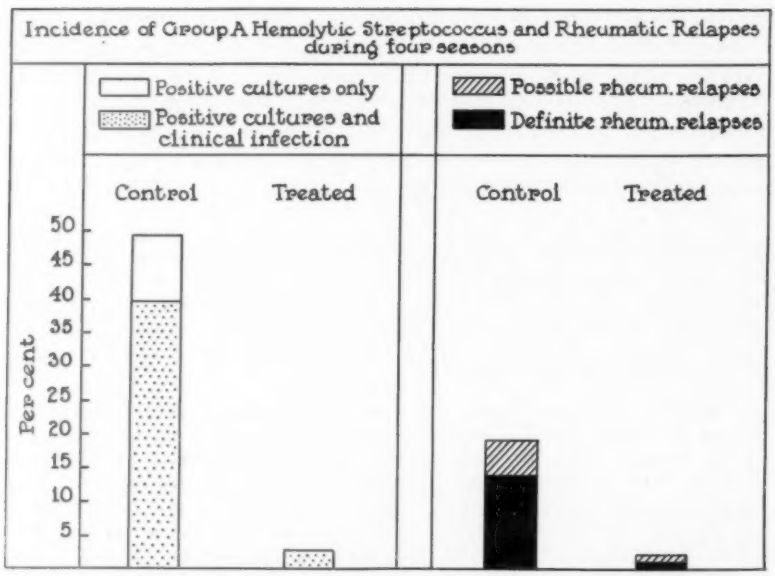

Fig. 4.—Incidence of Group A hemolytic streptococcus and rheumatic relapses in a control group of 101 patients observed for 138 patient-seasons and in eighty-eight patients given sulfanilamide prophylactically for 181 patient-seasons.

DISCUSSION

The incidence of rheumatic relapses is so variable from season to season and from individual to individual and is so greatly affected by environmental factors that evaluation of the results of any prophylactic procedure is always difficult. The fact that we used as controls a group of children of a younger age than those who were given sulfanilamide introduces a variable which must be taken into consideration in interpreting our results. That there is a difference from year

to year in the incidence of rheumatic fever is recognized. It has been the common impression in the children's cardiac clinics of New York City that the last few years have been so-called "light" rheumatic years.

Tables I and II assist in evaluating our results in terms of these two variables of age and season. In Table I patient-years of disease before treatment and patient-years when active disease occurred are recorded for each age and are compared to patient-years of disease and of activity while under treatment. In Table II the same data are recorded by age for the period before observation as control and for the period during observation as control.

TABLE I*

PATIENT-YEARS OF DISEASE AND OF RHEUMATIC ACTIVITY IN THE PATIENTS IN THE TREATED GROUP BEFORE AND DURING PROPHYLAXIS

AGE (YR.)	BEFORE PROPHYLAXIS		WHILE RECEIVING PROPHYLAXIS	
	PATIENT-YEARS OF DISEASE	PATIENT-YEARS OF RHEUMATIC ACTIVITY	PATIENT-YEARS OF DISEASE	PATIENT-YEARS OF RHEUMATIC ACTIVITY
3	4	4		
4	14	11		
5	26	16		
6	33	16	1	
7	46	29	1	
8	53	30	1	
9	57	33	9	
10	63	34	8	
11	53	28	18	
12	43	27	25	1
13	32	19	32	2
14	16	10	30	2
15	10	3	22	1
16	8	2	11	
17	3	2	12	
18			8	
19			2	
20			1	
Total	461	264	181	6†
Percentage		57.2		3.3

*In Tables I and II a patient is considered to be rheumatic from the year of his first attack until the present time. He is also considered to have a patient-year of activity during any year in which he had signs of rheumatic activity. Thus a child whose first attack of rheumatic fever was at the age of 5 years and who had subsequent attacks at the ages of 7 and 9 years and was treated during his eleventh and twelfth years would be recorded in the column, "Patient-Years of Disease Before Prophylaxis," in years 5 through 10; in the column, "Patient-Years of Rheumatic Activity Before Prophylaxis," in years 5, 7 and 9; and in the column, "Patient-Years of Disease While Receiving Prophylaxis," in years 11 and 12. All patients in the treated group are recorded in a similar manner. Thus all the children who had the disease at 4 years of age were active during that year; nineteen of thirty-two children observed at 13 years of age had active disease at that age, etc.

†Two patients who relapsed within first two weeks of taking sulfanilamide are included here.

It is seen (Table I) that in the treated group even in the adolescent years a great deal of active infection had taken place before sulfanilamide prophylaxis was begun and that 57 per cent of the total number of years of rheumatic disease in this group had been years in which rheumatic activity occurred. On the other hand, in the control group (Table II) only 40 per cent of the years of disease had been years in

TABLE II*

PATIENT-YEARS OF DISEASE AND OF RHEUMATIC ACTIVITY IN THE CONTROL GROUP BEFORE AND DURING OBSERVATION AS CONTROL

AGE	BEFORE OBSERVATION AS CONTROL		UNDER OBSERVATION AS CONTROL			
	PATIENT-YEARS OF DISEASE	PATIENT-YEARS OF RHEUMATIC ACTIVITY	PATIENT-YEARS OF DISEASE	PATIENT-YEARS OF RHEUMATIC ACTIVITY		
				DEFINITE	POSSIBLE	TOTAL
2	2	2				
3	4	2				
4	18	15				
5	33	19				
6	44	24	2			
7	57	29	4		2	2
8	68	25	2			
9	70	28	13	2	1	3
10	63	23	22	5	1	6
11	52	16	21	3	2	5
12	39	13	25	4	3	7
13	27	6	19		1	1
14	17	4	12	2	1	3
15	13	3	6			
16	6		6	2		2
17	3	1	3		1	1
18	2		1			1
19	1		1			1
20			1	1		1
Total	519	210	138	19	12	31†
Percentage active		40.4				22.4

*See footnote *, Table I.

†The discrepancy between this figure and that reported under results in the text is accounted for by the fact that in the above table 4 patient-seasons of continuing rheumatic activity and the one case of subacute bacterial endocarditis are included.

which active infection was present. There is a striking contrast between the incidence of active disease during the years before sulfanilamide was given and during the years in which the treated group received the drug.

As a possible indication of a decreased severity of the disease during the period of the study, Table II shows the number of recurrences in the control period as considerably fewer than in the same group of children during their years of disease before the study was undertaken. In any study of this sort there is the possibility that the intensive follow-up in itself exerts a favorable influence on the health of the children. This may be a further explanation of the difference in the amount of active disease in the control group before observation as control and during the subsequent control period.

Table II also demonstrates the fallacy of considering as control the expected incidence of recurrences based on the previous history of the patients treated. On this basis one would have expected recurrences in 40 per cent of those in the control group instead of the actual incidence of 22 per cent recurrences.* If the same factors which reduced

*The discrepancy between this figure of 22 per cent and that of the 19 per cent recurrences reported under results is due to the fact that in Table II the four patient-seasons of continuing rheumatic activity and the one case of subacute bacterial endocarditis are included in the column, "Patient-Years of Rheumatic Activity (Possible)."

the percentage of relapses in the control series from 40 to 22 may be assumed to have operated in the treated group, the expected incidence of recurrences in this group would have been 31 per cent rather than the 57 per cent of the pretreatment years of disease. The contrast between 31 per cent as an expected incidence and the 3.3 per cent which actually occurred is still striking and is even more so if one considers only the two children, who, in our opinion, represent definite failure of the drug to protect.

RESULTS AS REPORTED BY OTHER OBSERVERS

In order to reach definite conclusions as to the value of sulfanilamide in preventing relapses in rheumatic subjects and as to its safety when administered over long periods of time, it is essential that the results of all studies be pooled.

Table III gives the figures from all reports to date. There have been a total of ten definite rheumatic relapses in 706 patient-seasons of treatment, or an incidence of 1.1 per cent. Compared to this there were 116 major recurrences (18.1 per cent) among 639 patient-seasons of control observation, in addition to twenty-five minor or possible relapses. The uniformity of the results in all the reported series is striking and leaves little room for doubt that sulfonamide prophylaxis was the determining factor in the reduction of the incidence of rheumatic recurrences.

TABLE III

SUMMARY OF REPORTED RESULTS

	PATIENT-SEASONS CONTROL	RHEUMATIC RELAPSES	PATIENT-SEASONS TREATED	RHEUMATIC RELAPSES
Thomas and associates[1, 6, 7]	150	15	114	2
Coburn and Moore[2, 8]	146	31	188	1
Stowell and Button[4]	14	4	7*	0
Hansen, Platou, and Dwan[9]	46	14	78	1
Kuttner and Reyersbach[3]	104	28	108	1
Chandler and Taussig[10]	41	5	41	1
Present study	138	19	181	4†
			717	
Overlap of Kuttner and Reyersbach's study and the present study			−11	
Total	639	116	706	10
Percentage recurrence		18.1		1.4

*Preliminary study during one season only. During the following winter thirty-two children were started on the drug, but because of a fatal agranulocytosis occurring in a 12-year-old boy, the study was discontinued before any conclusions could be drawn.

†Two deaths from congestive heart failure included, in addition to the two definite relapses.

Toxic manifestations (Table IV) are more difficult to evaluate, because of the wide variation in their reported incidence by different observers. In this series there has been no reaction of sufficient severity to cause us to discontinue the drug permanently. On the other

hand, Stowell and Button[4] had already discontinued it in almost 30 per cent of the patients who had begun taking the drug before the occurrence of their case of fatal agranulocytosis caused them to abandon the study entirely.

TABLE IV

REPORTED TOXIC REACTIONS

	NUMBER STARTING THE DRUG	DRUG STOPPED	PERCENTAGE STOPPED	DOSAGE (GM.)
Thomas and associates[1, 6, 7]	55	2	3.6	1.0 to 1.3
Kuttner and Reyersbach[3]	100	15	15.0	1.0 to 2.0
Hansen, Platou, and Dwan[9]	53	1	1.8	1.3 to 3.0
Stowell and Button[4]	46	13	28.0	1.5 to 2.0
Chandler and Taussig[10]	39	5	13.0	0.6 to 1.5
Coburn and Moore[2, 8]			10.0	2.0 to 3.0
Present study	88	0	0	1.0 to 2.0
Total	381	36	9.4	

Among 381 patients who started taking the drug, thirty-six, or 9.4 per cent, discontinued it because of toxic reactions. Although the dosage given was perhaps somewhat smaller in the clinics reporting the fewest reactions to the drug, the difference does not seem to be great enough to account for the marked difference in the number of reactions reported. One must therefore conclude that the interpretation of what constitutes toxic reactions is an important factor in their incidence. While caution in the administration of a toxic drug is essential, it is possible that overcaution has led to discontinuance of sulfanilamide in some instances unnecessarily. The important fact is that only one of the many patients treated had a fatal reaction. Unfortunate as this was, it is hard to believe that among these same patients there would not have been many more than one death from rheumatic fever had the drug not been given.

PROBLEMS OF ADMINISTRATION OF CHEMOPROPHYLAXIS

We believe that there is now sufficient evidence to justify the use of a sulfonamide drug to prevent relapses in rheumatic children. If, however, such a drug is to be used routinely, a number of questions will arise which should be considered in advance. On some of these we have an opinion based on experience. Others can be answered only in the light of further study.

Selection of Cases.—If the development of a crippling degree of heart disease is to be prevented, then, obviously, young patients with a history of only one attack of rheumatic fever and with no evidence of heart disease are the ideal candidates for treatment. A fair proportion, however, of such children are never going to develop signs of permanent cardiac damage. We are therefore inclined to consider as candidates for prophylaxis children who have had two or more definite attacks of rheumatic fever with or without clear-cut evidence of

cardiac involvement or children whose one attack was accompanied by an acute carditis.

We believe also that children with long rheumatic histories and severe cardiac involvement should be considered candidates for prophylaxis, even although, as our results have shown, one cannot expect 100 per cent success in such cases. Considerable regression of cardiac signs may take place in children with advanced heart disease if they can be kept free of recurrent rheumatic fever for a number of years. Such patients are most easily kept under close observation and are most likely to cooperate in taking the drug regularly.

The question will arise as to whether chorea alone should be considered an indication for prophylaxis. In the reported studies children with chorea have been excluded unless they have shown other rheumatic manifestations. In the present study six children were in-

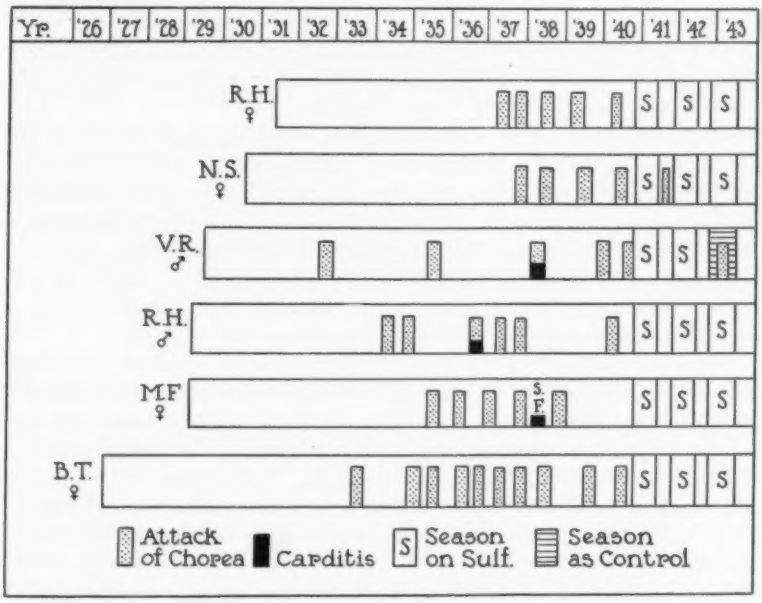

Fig. 5.—The histories of six children whose predominant rheumatic manifestation was chorea.

cluded in whom chorea was the predominant manifestation (Fig. 5). In three of these it was the only manifestation. Two of the other three had questionable active carditis accompanying one attack of chorea, and one had mild carditis with an attack of scarlet fever. None of these children had cardiac enlargement, although two had rather loud apical systolic murmurs. They had had from four to ten attacks of chorea each, the last occurring in five of the children less than a year before taking the drug.

These children were included with little expectation that the sequence of their bouts of chorea would be interrupted. To our surprise no attack of chorea occurred while any of these children were taking sulfanilamide. N. S. developed a mild attack in the summer after her first season on the drug. V. R., a boy who remained well during the two seasons he took the drug, developed chorea in March, 1943, when he was being followed in the control series. This attack occurred two months following a Group A hemolytic streptococcal pharyngitis.

It is true that the oldest patient, who had had ten attacks of chorea, was 15 years of age, the age at which the incidence of chorea is declining rapidly. However, she had had an attack at 13 years and another only a few months before starting the drug. The boy who relapsed during his final year as control was 14 years old.

Of course, no conclusions can be drawn on the basis of these six cases. However, it would seem that more of this type might be given a trial on the drug instead of being carefully and specifically excluded from groups being studied. It is possible, furthermore, that added light may be thrown on the relation of chorea to other rheumatic manifestations by such a study.

The Administration of Sulfanilamide to Patients With Recently Active Rheumatic Fever.—The question arises as to how soon a child convalescing from active rheumatic fever can be safely given the drug. It has been reported[11, 12] that sulfanilamide given during an attack of acute rheumatic fever not only fails to benefit the patient, but may even make him worse. It has also been suggested that the drug administered during the subsiding phase of an attack may precipitate a recrudescence.

Three children were started on the drug in the fall within one month and seven more within two months of the last sign of activity, usually an elevated erythrocyte sedimentation rate. Two of these ten developed signs of active rheumatic fever within two weeks of starting the drug. In three a transient rise in the erythrocyte sedimentation rate for one week only occurred without other evidence of rheumatic activity. The remaining five developed no signs or symptoms of rheumatic reactivation.

The two children who developed rheumatic symptoms were continued on their daily doses of sulfanilamide. Their attacks were mild and their signs of activity diminished gradually over a period of weeks. In one the erythrocyte sedimentation rate remained elevated for eight weeks, with tachycardia and gallop persisting for a few weeks longer. In the other the erythrocyte sedimentation rate became normal after six weeks and the heart rate in two months.

Therefore, although we have preferred that a child be without laboratory and clinical evidence of active rheumatic infection for from two to three months before starting the drug, we should not hesitate to use it earlier in the case of a child whose rheumatic attack subsided

in the late fall. In such a case we believe the danger of precipitating a relapse by the administration of the drug is less than that of allowing the child to enter the streptococcus season unprotected.

Choice of Drug.—It has been a great advantage in evaluating the results of all studies to date that one drug, sulfanilamide, has been used almost exclusively. It would now seem desirable, however, to give a trial to other sulfonamides which are known to be equally effective in streptococcal infections and which are, in general, less toxic. Because of its slower elimination, sulfadiazine in small doses would probably maintain a more constant blood level, even if the interval between doses were relatively long. Watson and associates[13] have recently reported the control of an epidemic of scarlet fever in a United States Naval Station by the prophylactic use of 1 Gm. of sulfadiazine daily. Two-thirds of the men receiving the drug were given it in only one dose a day. Among several thousand men treated, only three had rashes due to sulfadiazine, and no major toxic reactions occurred during the few weeks the drug was taken.

Until some new and less toxic drug is developed, it would appear that sulfadiazine in doses of not more than .5 Gm. a day for small children and 1 Gm. for larger children and adolescents might be substituted for sulfanilamide in the prophylaxis of rheumatic fever. No real effort has been made to study the minimal effective dose of these drugs for this purpose. It may prove that even smaller doses of sulfadiazine are effective in preventing streptococcal infections.

Laboratory Follow-Up.—The most serious problem to be considered before undertaking the prolonged administration of a sulfonamide is, of course, that of the laboratory work essential to safeguard its administration. It is obvious that the drug will be difficult to administer in a large clinic if several special technicians are necessary to ensure its safety. Bacteriologic studies can be omitted. An occasional blood or urine drug level determination would be advisable in order to check on the child's reliability in taking the drug. No case of hemolytic anemia has been reported on the small dose recommended. Those which have been reported on much larger therapeutic doses have been reversible, and the hemoglobin and red blood cell count have risen promptly when the drug was discontinued. It would, therefore, seem not too hazardous to suggest that careful clinical examination might be substituted for a routine hemoglobin and red blood cell determination and that this be reserved for those who look suddenly pale.

This leaves only the important question of the white blood cell count and differential to be considered. During our fourth season we continued to do routine total counts at every clinic visit, but a chamber differential was done only if the total count was below 5,000. If the chamber differential count revealed polymorphonuclear cells of less than 60 per cent a stained smear was studied. It is interesting to speculate

as to what might have happened to the total and granulocyte counts of those children in whom the drug was temporarily discontinued because of a leucopenia had they been left on the drug. The one case of fatal agranulocytosis reported by Stowell and Button[4] occurred in a child who had been having semiweekly blood counts. Thus even this precaution, which has been considered sufficient by all users of the drug, did not prevent a fatal reaction, which occurred suddenly, and without a warning gradual reduction in leucocytes.

If it is conceded that sulfonamide prophylaxis is effective in inactive rheumatic patients and if clinics hesitate to employ this effective agent because of fear of toxic reactions or because of inability to provide the needed laboratory assistance, then obviously some children are going to die of rheumatic relapses who might otherwise survive for many years.

Duration of Sulfonamide Prophylaxis.—Having instituted sulfonamide prophylaxis, shall the drug be given the year around or during the winter and spring months only? Since most of the toxic reactions which have been reported have occurred during the first few weeks of the administration of sulfanilamide, the burden in clinics where large numbers are seen is heaviest during that period. While relatively few rheumatic relapses occur during the summer, a certain number of attacks do occur during that season. In the present study there were three relapses that developed between sulfanilamide seasons. Therefore, since it has not yet been shown that the prolonged administration of the drug has any deleterious effect, it would seem simpler to keep a child who is tolerating the drug well on it continuously rather than to stop it during the summer. This point needs further investigation.

There are several questions which require further study before a final answer can be given. For how many years should prophylaxis be continued? Present knowledge of the course of the disease would make it reasonable to continue it until a child is well into adolescence or for five years after the onset of his disease.

Only careful study and prolonged observation of large numbers of rheumatic children who have received sulfonamide prophylaxis will enable us to reach any conclusion on its effect on the subsequent course of the disease and on the subsequent susceptibility to hemolytic streptococcus infections in individuals formerly protected against them.

SUMMARY AND CONCLUSIONS

1. Eighty-eight children and adolescents with quiescent rheumatic disease were given from 1 to 2 Gm. of sulfanilamide daily throughout the winter and spring months for a total of 181 patient-seasons.

2. One hundred and one rheumatic children were observed as controls for 138 patient-seasons.

3. In the group receiving sulfanilamide, in some cases for as many as four seasons, toxic drug reactions were minimal. The drug was not discontinued permanently in any case for such a reaction.

4. During the period of the study there were in the control group 54 Group A hemolytic streptococcal infections, an incidence of 39 per cent. There were nineteen definite major rheumatic relapses (with two deaths) and seven mild or possible relapses. In three children the rheumatic process remained active throughout the period of observation, and there was one death from subacute bacterial endocarditis.

5. In contrast to this, only five hemolytic streptococcal infections occurred in the group of children receiving sulfanilamide prophylaxis, an incidence of 2.7 per cent. Two children, or 1.1 per cent of the patient-seasons of prophylaxis, developed definite rheumatic relapses while taking the drug regularly. Two other children with severely damaged hearts died of congestive failure without evidence of streptococcal infection or active rheumatic disease. Two children with recently active rheumatic fever showed signs of increasing rheumatic activity within two weeks of starting the drug. The remainder of this group of children remained free of streptococcal infection and rheumatic relapses.

6. Some of the problems of the administration of a sulfonamide prophylactically have been discussed.

7. It is our opinion, based on this study and reports in the literature, that the effectiveness of sulfonamide prophylaxis in quiescent rheumatic fever is established and that it should be applied more widely among groups of highly susceptible individuals.

We wish to express our appreciation to Dr. Homer Swift and to his associates, Dr. Robert F. Watson and Dr. Armine T. Wilson, for their invaluable advice and cooperation in this study.

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RHEUMATIC FEVER AND RHEUMATIC HEART DISEASE IN LOS ANGELES CHILDREN

A Hospital Study

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MANY writers in recent years have emphasized the relatively low incidence of rheumatic fever in warm climates. Coburn,[1] after transporting ten patients with severe rheumatic heart disease from New York City to San Juan, Puerto Rico, in 1929, concluded that "the rheumatic process, severe in New York, subsided during three months in the tropics, disappeared clinically during six months in the tropics, and evidenced itself with sudden reappearance of symptoms in some instances shortly after the return of these patients to New York. These observations strongly suggest that the rheumatic state was influenced by the change in environment." Jones and associates,[2] in 1930, in an attempt to check this effect of climate upon the activity of the disease, transported a group of rheumatic fever patients from Boston to Miami Beach, Fla. The conclusions drawn from this experiment were as follows: "Rheumatic fever and rheumatic heart disease subjects living in such a climate (i.e., Florida) seem to be protected to some extent, but not invariably . . . the patients with mild rheumatic heart disease or early mild rheumatic fever seemed to improve more strikingly. . . . Caution is expressed with regard to the unquestionable value of transportation to a subtropical climate." Nichol,[3] in 1936, stated that "available data indicate that there is a definite inequality in the distribution of rheumatic fever and rheumatic heart disease in the United States, the amount being much less in Southern states." Seegal, Seegal, and Jost,[4] in an analysis of hospital questionnaires, found that the frequency of rheumatic fever diminished from latitude region 50-45 degrees to latitude region 34-29 degrees.

Although statistics have been presented from many population centers in the United States concerning the incidence of rheumatic heart disease, no recent data are available for the Los Angeles area. Figures for this city are those presented by Faulkner and White,[5] who, in 1924, found that the incidence of rheumatic fever and chorea at the Los Angeles General Hospital was 0.44 per cent, or one case in every 227 admissions. This was based on a study of the adult medical admissions (13,614) from July, 1921, to July, 1923. More recent statistics are those of Hyland,[6] which were, however, based on a study of 500 consecutive autopsies on children at the Los Angeles Children's Hospital. Fifteen cases of acquired heart disease were encountered in this series (3 per cent).

The present report is an attempt to determine the prevalence of rheumatic fever and rheumatic heart disease in Los Angeles children on the basis of an analysis of the records at the Los Angeles General Hospital of all children up to and including the age of 13 years admitted to the pediatric wards and the Contagious Disease Unit in the five-year period from July 1, 1936, to June 30, 1941. Measuring the local prevalence of rheumatic fever by the number of hospital admissions may not be entirely dependable, but it should present a fairly good indication of the incidence and severity of the disease in the low income group cared for at the Los Angeles General Hospital, located in this community of latitude 34 north. It is obvious that the incidence as determined by hospital records does not portray the picture seen in the average pediatric practice or in a cross section of the public schools.

It is to be noted that the approach to the subject in this study differs from that of most published hospital figures in that only those admitted to the pediatric wards are herein considered, in contrast to the usual practice of including total medical or total hospital admissions. In the light of this approach, the role of the pediatrician in the program for control of rheumatic fever assumes greater importance.

Criteria.—The diagnosis of rheumatic fever and rheumatic heart disease was accepted from the records except in those cases where there was reasonable doubt. These latter were excluded. The criteria for diagnosis were as follows:

A. Clinical
   1. Polyarthritis
   2. Fever
   3. Cardiac findings
   4. Chorea
   5. Subcutaneous nodules
   6. Skin manifestations

B. Laboratory
   1. Blood count
   2. Sedimentation rate
   3. Electrocardiogram
   4. Orthocardiogram and x-ray

In analyzing the case histories the following, information was elicited:

1. Total number admitted to pediatric wards

Children 13 years and under admitted to the pediatric wards and the Contagious Disease unit at the Los Angeles General Hospital (Medical Division) from July 1, 1936, to June 30, 1941. This does not include the newborn service.

2. Total cases of rheumatic fever and rheumatic heart disease
3. Those patients with multiple admissions
4. Total admissions for rheumatic heart disease, active and inactive

5. Total admissions for chorea
   a. With rheumatic heart disease
   b. Without rheumatic heart disease
6. Seasonal and yearly incidence
7. Age at admission
8. Sex
9. Race
10. Familial incidence
11. Place of birth
12. Length of time in Los Angeles before onset
13. Incidence of prior disease
    a. Sore throat
    b. Scarlet fever
    c. Nephritis
14. Complications
15. Previous tonsillectomies
16. Length of stay in hospital
17. Deaths

Admissions.—In Table I are given the figures for the total number of admissions by the year and the admissions for rheumatic disease. These figures are shown graphically in Fig. 1.

TABLE I

YEAR	PEDIATRIC ADMISSIONS	CONTAGIOUS DISEASE ADMISSIONS	TOTAL ADMISSIONS	RHEUMATIC FEVER RHEUMATIC HEART DISEASE, CHOREA ADMISSIONS
1936-1937	2,744	2,051	4,795	45
1937-1938	2,312	2,216	4,528	32
1938-1939	2,550	1,991	4,541	38
1939-1940	2,372	1,472	3,844	38
1940-1941	3,157	1,672	4,829	20
Totals	13,135	9,402	22,537	173

The 173 admissions for rheumatic fever, chorea, and rheumatic heart disease were accounted for by 157 patients, sixteen, or approximately 10 per cent, of whom had more than one admission. This represents a case incidence of 0.69 per cent, or one case in 143.5 admissions to the pediatric wards and to the Contagious Disease Hospital. One hundred twelve of the total 173 admissions entered with active rheumatic heart disease, while twenty-one were inactive. The diagnosis of acute rheumatic fever was made in seventy-eight instances.

There were twenty-seven admissions for chorea during the period of study (15.6 per cent), seventeen associated with rheumatic heart disease (9.8 per cent) and ten without (5.8 per cent). One child with rheumatic heart disease was admitted twice with chorea. This incidence of chorea is less than one-third that found by Jones and Bland[7] in 1935

in a New England survey, three-fifths of that noted by Mackie[8] in New York in 1926, one-half of that found by Bilderback and Overstreet[9] in Oregon in 1938, and about three-fifths of that found by Christie[10] in San Francisco in 1936.

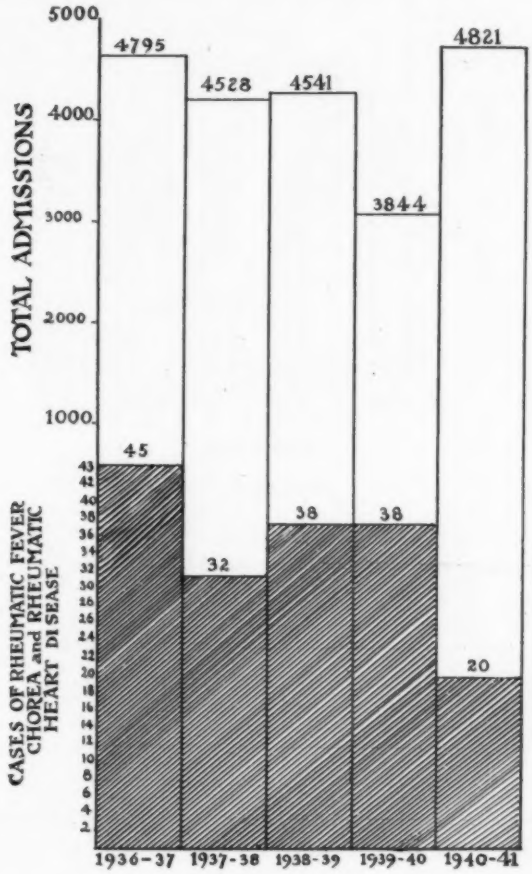

Fig. 1.

Seasonal and Yearly Incidence.—Except for a moderately lower incidence during the months of January, May, June, and August, the seasonal incidence of the disease remained relatively constant (Fig. 2).

Thus it may be stated that in the group studied here there was no particular season for rheumatic fever as is noted in other communities. A sharp decline in incidence, both relative and absolute, is manifested in 1940-1941 (see Fig. 1), the explanation for which remains obscure. To be considered as a possible reason for this lower incidence is the decrease in unemployment during this period and the subsequent improved

standards of living. Of questionable significance is the variation in the incidence of scarlet fever during this same period, as demonstrated in Table II.

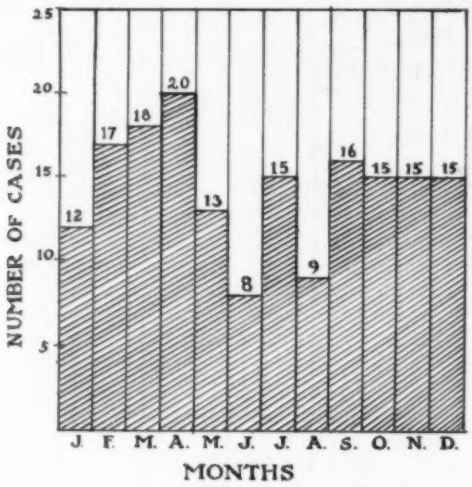

Fig. 2.

TABLE II

YEAR	CASES OF SCARLET FEVER ADMITTED	CASES OF RHEUMATIC FEVER, CHOREA AND RHEUMATIC HEART DISEASE
1936-1937	542	45
1937-1938	582	32
1938-1939	608	38
1939-1940	437	38
1940-1941	396	20

TABLE III*

	TEMP. (MEAN)						AVERAGE TEMPERATURE FOR 64 YEARS
	1936	1937	1938	1939	1940	1941	
Jan.	59.4	47.8	61.2	57.0	59.8	57.6	55.4
Feb.	56.5	55.3	56.8	54.0	59.4	58.8	56.3
March	59.0	58.5	57.8	56.8	61.3	60.6	58.1
April	60.6	62.3	61.6	63.0	62.8	59.1	60.2
May	65.6	64.0	63.3	63.6	66.0	67.9	62.7
June	69.0	67.8	65.4	66.9	66.0	66.0	66.5
July	74.1	71.4	69.6	70.6	70.6	70.7	70.5
Aug.	73.4	71.6	73.9	73.0	70.6	71.2	71.4
Sept.	70.5	72.4	74.0	76.6	69.6	68.0	69.7
Oct.	67.4	67.6	66.7	71.6	68.3	66.0	65.7
Nov.	67.8	61.7	62.7	67.1	63.4	65.4	62.0
Dec.	58.6	61.9	62.3	64.2	61.2	57.0	57.2
Average	65.2	63.5	64.6	65.4	64.9	64.0	63.0

*Taken from the Annual Meteorological Summary compiled by the Weather Bureau of the United States Department of Agriculture.

Data on Climate.—Since temperature, rainfall, and relative humidity have been considered vital factors in the incidence of rheumatic infection, we believe it pertinent to present Tables III, IV, and V, published by the Department of Agriculture.* Perusal of these figures for the

TABLE IV*

	PRECIPITATION IN INCHES						AVERAGE PRECIPITATION FOR 64 YEARS
	1936	1937	1938	1939	1940	1941	
Jan.	0.51	1.99	1.63	2.96	4.33	2.21	3.08
Feb.	7.25	7.87	9.81	1.13	5.43	12.42	3.35
March	1.34	4.04	7.94	1.44	1.55	8.14	2.75
April	0.95	0.24	0.48	0.24	1.61	2.67	1.06
May	0.00	0.28	0.02	0.02	0.02	T	0.39
June	0.20	0.00	T	T	T	T	0.08
July	0.01	0.00	0.00	T	T	T	0.01
Aug.	0.02	0.00	T	0.01	0.00	0.04	0.02
Sept.	0.03	0.00	0.01	5.67	0.01	0.00	0.23
Oct.	1.25	0.01	0.01	0.13	1.47	1.53	0.65
Nov.	0.05	0.00	T	0.08	0.34	0.05	1.09
Dec.	6.63	3.54	7.26	0.38	5.50	4.22	2.86
Total	18.24	17.97	27.16	12.06	20.26	31.28	15.57

*See footnote, Table III.
T, Trace.

TABLE V*

	HUMIDITY (RELATIVE % AT 12 NOON)						AVERAGE HUMIDITY FOR 26 YEARS
	1936	1937	1938	1939	1940	1941	
Jan.	38	42	37	48	54	51	45
Feb.	57	50	52	40	49	62	51
March	52	47	46	55	46	51	49
April	56	44	50	52	52	53	52
May	50	59	52	55	52	47	56
June	53	57	61	54	59	59	57
July	50	53	56	54	47	52	54
Aug.	52	54	47	52	53	54	54
Sept.	49	49	45	42	47	49	51
Oct.	47	50	42	32	45	44	47
Nov.	26	54	24	38	31	32	37
Dec.	44	42	40	37	45	53	44
Average	48	50	46	47	48	51	50

*See footnote, Table III.

period studied permits no correlation between any of the foregoing factors and the incidence of the disease in this area. Thus, in 1940-1941, when the incidence of rheumatic disease was less than one-half that in 1936-1937, the precipitation was considerably heavier than in the latter year, and the temperature and humidity were approximately the same. To carry this further, a comparison is made in Table VI of the figures for July and December, during which months the incidence of the disease was the same.

Race.—Of the 157 patients, eighty-two were white, sixty-one Mexican, and fourteen Negro. The high percentage of Mexican children is in keeping with the relatively high percentage of Mexican patients ad-

TABLE VI

	TEMP. (MEAN)	PRECIPITATION	RELATIVE HUMIDITY	NUMBER OF CASES OF RHEUMATIC HEART DISEASE
July	70.5° F.	0.01	54	15
December	57.2° F.	2.86	44	15

mitted to the General Hospital and does not necessarily denote an abnormally high incidence of the disease in these people. It may reflect, however, their low standard of living.

Age.—The youngest patients in the present series were 4 years old, six belonging to this age group. Over one-half of the children were above the age of 10 years when admitted to the hospital. Sixty-nine were under this age. (See Fig. 3.)

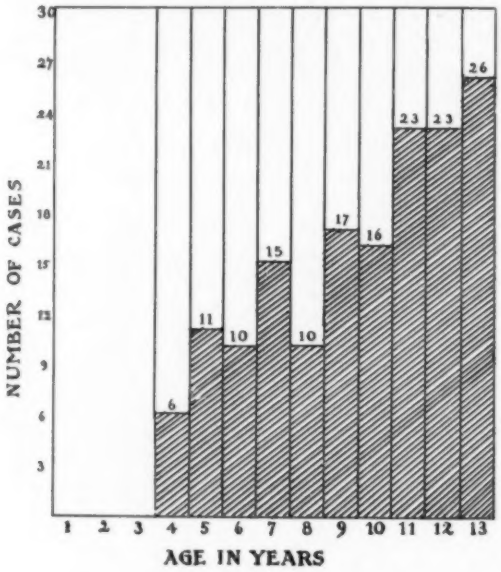

Fig. 3.

Sex.—Analysis as to sex revealed no essential difference in this series, seventy-seven being boys and eighty girls. This is in contrast to the commonly reported preponderance in females.

Family Incidence.—In only fourteen children (8.9 per cent) was a familial incidence noted. The relationship is shown in Table VII.

Although the recent trend has been toward the concept of rheumatic fever as a contagious disease, occurring frequently in epidemic form, our experience in this community based on this survey does not warrant such a conclusion. In only one instance was it noted that more than

TABLE VII

RELATION	CASES
Mother	4
Father	3
Uncle	2
Sister	3
Brother	1
Aunt	1

one member of a family was afflicted with the disease at the same time. This occurred when a 13-year-old Negro female and her 4-year-old brother were admitted to the hospital on the same day, both having active rheumatic heart disease on this, their first admission to the hospital. The familial incidence noted in Table VII, except for these two siblings, refers to old inactive cases, bearing no apparent relationship to the acute episodes of these children.

Place of Birth and Length of Time in Los Angeles Before Onset of Disease.—In Table VIII we have listed the places of birth of all children born outside of Southern California with their lengths of stay in this community before the onset of the disease. In all, fifty-four children (34.3 per cent) came from different localities, with the length of stay in Los Angeles of these patients varying from two weeks to twelve years. Thus, 103 (65.7 per cent) of these patients were born and raised in this area. It is of interest that thirty-one of the fifty-four children from outside Southern California came from Southern states, with eighteen from Arizona, Missouri, and Texas. The only New England state represented was Massachusetts, which contributed three cases to the total, two of the patients having been in Los Angeles three and ten months, re-

TABLE VIII

PLACE OF BIRTH	NUMBER	LENGTH IN LOS ANGELES
Arizona	5	3, 7, 9, 3, and 4 yr.
Arkansas	3	9, 11, and ? yr.
Colorado	3	5, 3, and 6 yr.
Denmark	1	7 yr.
Iowa	1	5 yr.
Illinois	2	2, and 9 yr.
Kansas	1	7 yr.
Minnesota	2	?, and ?
Missouri	5	3, ?, 10, 4, and 6 yr.
Michigan	1	4 mo.
Massachusetts	3	10 mo., 3 mo., and 5 yr.
Maryland	1	4 yr.
Mexico	2	11, and 12 yr.
New Mexico	2	2 wk., and ?
New Jersey	1	?
New York	4	2 wk., 3, 3, and 4 yr.
Ohio	1	9 yr.
Oklahoma	4	4, 4, 6 yr.
Pennsylvania	1	8 yr.
Texas	8	1, ?, 1, 11, 1, 1, 3, and 6 yr.
Utah	1	2 wk.
Virginia	1	3 yr.
Washington	1	10 yr.

spectively. One child from New Mexico, one from New York, and one from Utah were in the community only two weeks before the onset of the disease.

The fact that two-thirds of the children in this group were born and raised in Los Angeles would certainly tend to discredit the commonly held belief that the majority of patients with rheumatic disease encountered here are migrants from eastern or northern centers. To emphasize this point further, forty-two of the fifty-four children who were born out of this state lived in Los Angeles over one year before the onset of the disease.

Incidence of Prior Disease.—Fifty-six patients entered the hospital with a history of recent "sore throat," a percentage of 32.3 for all admissions. Jones and Mote,[11] in Boston, found that 66 per cent of their patients had "sore throats" prior to the onset of rheumatic fever.

Only one patient in the present series had nephritis prior to the onset of rheumatic fever, while twenty-four (15.3 per cent) had a history of scarlet fever. These findings are consistent with those reported in similar studies elsewhere.

Tonsillectomies.—Of the 157 patients with rheumatic fever, chorea, and rheumatic heart disease, forty-seven (29.9 per cent) gave a history of tonsillectomy. Forty-two (26.7 per cent) had this surgery performed prior to the first attack, while five had the operation subsequent to the onset of the disease. One patient had his tonsillectomy three days before the first attack of rheumatic fever. Of the fifteen fatal cases in our series, it will be noted that twelve of the patients had not had adenotonsillectomies.

Complications.—Seventy-nine patients entered the hospital with, or developed while on the ward, one or more complications (Table IX).

TABLE IX

COMPLICATIONS

Upper respiratory infection	17
Epistaxis	15
Pneumonia	12
Erythema multiformi	5
Pulmonary tuberculosis	4
Appendicitis	3
Subcutaneous nodules	3
Scarlet fever	3
Cervical adenitis	3
Subacute bacterial endocarditis	2
Alopecia	1
Poliomyelitis	1
Hypertension	1
Schönlein's purpura	1
Mental deficiency	1
Measles	1
Mumps	1
Epilepsy	1
Congenital syphilis	1
Tetanus	1
Erythema nodosum	1
Osteomyelitis	1

Seventeen of the children had upper respiratory infections during the course of the disease, while twelve entered with pneumonia. We have listed epistaxis as a complication, fifteen patients presenting this finding. On three children, subcutaneous nodules were found, while five manifested erythema multiformi (rheumatic) and one erythema nodosum. It is of interest that only two patients developed subacute bacterial endocarditis during this five-year period of observation.

Of rather frequent occurrence during the onset of the disease was the complaint of abdominal pain. In several patients appendicitis was considered likely on entrance. In one patient who later developed a pericardial effusion, an emergency exploratory laparotomy revealed no intra-abdominal pathology.

Length of Stay in Hospital.—The 157 patients spent a total of 5,775 days in the hospital, an average of 36.6 days each. The longest stay was 186 days, while eighteen patients remained under observation less than one week. Twenty patients were in the hospital for more than fifty days.

Deaths.—Of the 157 patients, fifteen died during this five-year period of analysis, a mortality of 9.5 per cent. Summary of these cases is as follows:

1. A 13-year-old Mexican female entered the hospital Oct. 24, 1939, and died Nov. 12, 1939. This was the first admission. She had lived in Southern California all her life. The family history was negative, as was the past history, except for sore throats since the age of 10 years. The patient had not had a tonsillectomy. She had complicating pneumonia with pancarditis and passive congestion.

2. A 7-year-old Mexican boy entered July 27, 1939, and died Aug. 15, 1939. He had lived in California all his life. This was the first attack. The child had not had a tonsillectomy. He died after a laparotomy with acute rheumatic fever, endocarditis, and pericardial effusion.

3. A 7-year-old Mexican female entered the hospital Nov. 9, 1939, and died Nov. 17, 1939. She had lived in California all her life. This was the first attack. She had never had a tonsillectomy. The family and past histories were negative. The child died with decompensation and pericardial effusion; she had had chronic malnutrition.

4. A 12-year-old white female entered the hospital May 16, 1939, and died May 23, 1939. She had lived in Los Angeles all her life. This was the second attack. There was no relevant past history or family history. She had not had a tonsillectomy. The child died with pancarditis and decompensation.

5. A 6-year-old Mexican female entered the hospital May 3, 1939, and died May 25, 1939. She was born and raised in Los Angeles. The family history was negative. This was the second admission (first in 1936 with chorea and rheumatic heart disease). She had had a tonsillectomy at age of 6 years. The child died in exacerbation of rheumatic fever with subacute bacterial endocarditis.

6. A 4½-year-old white boy entered the hospital Jan. 6, 1939, and died Jan. 28, 1939. He had lived in Los Angeles all his life. This was the first attack. The past and family histories were negative. He had not had a tonsillectomy. He had rheumatic nodules and erythema multiformi. The child died with rheumatic pancarditis and passive congestion, with possible rheumatic pneumonia.

7. An 8-year-old Mexican female entered the hospital Feb. 6, 1939, and died Feb. 8, 1939. She had lived in Southern California all her life. This was the first attack. The family and past histories were negative. She had not had a tonsillectomy. The child died with chronic rheumatic endocarditis, acute osteomyelitis with staphylococcic septicemia, and abscesses in heart muscle and lung.

8. A 10-year-old Negro female entered the hospital March 23, 1939, and died March 25, 1939. She had lived in Los Angeles all her life. The first attack occurred one month prior, with the patient bedfast ever since. The past and family histories were negative. She had not had a tonsillectomy. The child died with pericarditis, decompensation, and bronchopneumonia.

9. A 13-year-old white female entered the hospital Oct. 28, 1939, and died Oct. 31, 1939. She had lived in Los Angeles all her life. An uncle had rheumatic fever. She had not had a tonsillectomy. The child had been in the hospital one and one-half years previously for three months; she had been taken out against consent while still having a septic fever. The child died with subacute bacterial endocarditis. She also had subcutaneous nodules.

10. A 10-year-old white female entered the hospital March 24, 1938, and died April 12, 1938. She was born in Virginia and had lived in Los Angeles three years. This was the first attack. She had had scarlet fever at the age of 8 months. A tonsillectomy was performed but the date is not known. The child died of pancarditis, decompensation, and pneumonia.

11. A 6-year-old Negro male entered the hospital Nov. 13, 1937, and died Dec. 16, 1937. He was born and raised in Los Angeles. No tonsillectomy had been performed. This was the first attack. The child had had scarlet fever three weeks prior to onset. He died in congestive failure.

12. A 9-year-old white female entered the hospital Aug. 2, 1937, and died Aug. 4, 1937. She was born in Denmark and had lived in Los Angeles seven years. This was the second admission for rheumatic heart disease. She had not had a tonsillectomy. The child died in decompensation with auricular fibrillation.

13. A 13-year-old Mexican male was born and raised in Southern California. He entered the hospital Feb. 14, 1938, and died May 13, 1938. The disease was noted first in 1936 with one subsequent entry. The family history was negative. The child had not had a tonsillectomy. He died with pancarditis and decompensation.

14. A 12-year-old Mexican female entered the hospital Nov. 24, 1936, and died Dec. 1, 1936. She was born in Texas but had lived in Los Angeles since the age of 1 year. The first attack occurred in the early part of 1936. The child had had a tonsillectomy at the age of 8 years. She died with acute myocarditis and bronchopneumonia.

15. An 8-year-old white boy entered the hospital July 6, 1936, and died July 10, 1936. He was born in Texas and had lived in Los Angeles for only one year. He had had repeated attacks of the disease during the previous two years. No tonsillectomy had been performed. He died in congestive failure.

In summarizing, it is to be noted that seven of the deaths were in Mexican children, two in Negro, and six in white, that ten of the children were girls, and that the ages were equally divided below and above 10 years. Eleven of the fifteen children who died were born and raised in this community, while of the remaining four, three had lived in the city for two or more years. Two of the three children with subcutaneous nodules were in this death group. Eleven of the deaths were due to extensive myocardial damage with associated valvular lesions and decompensation. Two resulted from subacute bacterial endo-

carditis, and one from acute osteomyelitis and sepsis. One patient with pancarditis died following exploratory laparotomy.

SUMMARY

The cases of 157 patients with childhood rheumatic disease admitted to the Los Angeles County Hospital over a five-year period are analyzed and discussed. The following salient findings bear emphasis:

1. The relatively low incidence of rheumatic disease as determined by this survey is consistent with the findings of similar studies carried out in other cities with subtropical climates.

2. Chorea seems to occur with much less frequency in this area than in eastern population centers.

3. No significant variation in seasonal incidence is noted.

4. An analysis of temperature, rainfall, and relative humidity permits no correlation between these factors and the incidence of the disease in this area.

5. Familial incidence in this series is negligible.

6. Two-thirds of the patients were born and raised in this community. Almost 80 per cent of those born out of California lived in the Los Angeles area over one year before the onset of rheumatic disease. These facts would certainly tend to discredit the commonly held belief that the majority of the patients with rheumatic infection encountered here are migrants from eastern or northern states.

7. The mortality in this series was 9.5 per cent. Brief summaries are presented of all cases that proved fatal.

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RHEUMATIC PERICARDITIS WITH EFFUSION IN PATIENTS UNDER TWO YEARS OF AGE

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THESE two cases of pericarditis with effusion are reported not because of the rarity of the condition but because of its rarity in children of these ages. McIntosh and Wood[1] in 1935 reviewed all the admissions to the Boston Babies Hospital for twenty-five years and collected twenty-four cases of rheumatic infection in children below the age of 3 years. They reviewed the ages of the patients reported by other authors. Still[2] reported 1,027 cases of rheumatic infection with eight patients under 3 and none under 2 years of age. Findlay[3] reported 457 cases of rheumatic carditis with two patients between 1 and 2 years and three between 2 and 3 years. Smith and Sutton[4] reported 623 cases of rheumatic infection with three patients below 2 years of age. From a compilation of these figures, there were fifteen, or .7 per cent, occurring within the first three years of life. There were only five of the 2,107 cases of rheumatic infection in patients below 2 years of age, about 1 in 400, or less than .25 of 1 per cent.

McIntosh and Wood do not report the number of patients in age groups by years or months, simply those below 3 years of age. Of six of their patients on whom autopsies were performed, there were three below 2 years of age. Their conclusions were "the incidence of rheumatic infection in subjects under 3 years of age is probably greater than is generally recognized. The history, symptoms, and laboratory data in these cases are variable and the clinical picture is more often that of a general infection than that of a specific disease entity. The outstanding feature of rheumatic infection during the first three years of life is cardiac damage. In our series 96 per cent of the children exhibited either clinically or pathologically rheumatic heart disease."

Of their twenty-four patients there was a pericardial friction rub in eight, an incidence of 33 per cent. In two, pericardial fluid was aspirated.

By exclusion and by the course of the symptoms and findings, these two patients being reported are probably examples of rheumatic pericarditis. The onset of their symptoms agrees with the conclusions of McIntosh and Wood that rheumatic infection in the age group below 3 years is more often that of a general infection than that of a specific disease entity.

The first patient was seen about six years ago. The second patient was seen very recently.

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REPORT OF CASES

CASE 1.—B. K., 22 months of age, was examined at home by me for another physician, the family doctor. There was a history of fever and malaise of three days' duration. Rectal temperature was 102° F. The child did not appear ill and reacted violently to the examination. There was some edema and redness of the pharynx and slight cervical adenitis. The heart tones were muffled and the cardiac borders markedly enlarged both to the right and left. The left border was in the midaxillary line. The cardiohepatic angle was obtuse. The pulse was rapid and strong. There were no other findings; no joint involvement. Slight dyspnea occurred after struggling and crying during the examination. There were no other findings. The temperature remained elevated for three weeks and the child was kept in bed for six weeks. Enlarged cardiac borders and evidence of cardiac involvement existed for three weeks. The child has remained well and has had no rheumatic episodes. She has been seen a number of times since, when the family doctor was not available, as recently as a year ago. She has remained very well with no evidence of cardiac disability symptomatically or by physical examination.

CASE 2.—J. S., 8 months of age, had been under routine pediatric care since birth. She was first seen for this illness May 12, 1943, in the home because of a sudden rise in temperature to 104° F. at 10:00 A.M. When first seen at 4:30 P.M. the temperature had risen to 104.6° F. rectally. The only finding was an injected, slightly edematous pharynx. The baby did not appear particularly ill. The next morning the mother called to say that she was concerned because the temperature was 104° F. but that instead of being flushed, the baby was ashen in color. When seen at home the mother's descriptive term of ashen was not exaggerated. There was a marked hoarseness to the baby's cry and a slight, short cough after crying. There was considerable dyspnea. She was brought into the hospital for care and diagnosis. The provisional diagnosis was an early pneumonia.

At the hospital the following diagnostic procedures were carried out: throat culture, blood culture, x-ray of lungs and heart, blood cell count, and urinalysis.

The blood cultures were negative. The x-ray report showed the following: The hilar shadows and peribronchial markings were of soft density. The diaphragmatic outline was smooth. The peripheral areas were clear. The heart appeared large but was considered within the normal. Résumé: The chest was considered negative for pneumonia. Blood cultures were negative May 15 and May 20, 1943. Throat culture showed a moderate growth of *Staphylococcus aureus*, nonhemolytic streptococcus, and *Streptococcus viridans*. The blood count showed the following: red blood cells, 3,860,000; hemoglobin, 9.9 Gm.; white blood cells, 22,250, with 58 per cent neutrophiles, 40 per cent lymphocytes, and 2 per cent monocytes. The urinalysis was negative. Management and Treatment: The child was given sulfadiazine, 5 gr. every four hours for three days; fourth and fifth day, 5 gr. every six hours. There was no indication of response to this drug.

There was a gradual increase in dyspnea and on the morning of May 18, the sixth day of the illness, it became apparent from the marked increase in cardiac dullness and the obtuse angle of the cardiohepatic angle of dullness that a pericarditis existed. X-ray of the heart shadow confirmed this. The x-ray report showed the following: The cardiac shadow was seen to be of marked increase in size. It was somewhat globular in shape. On comparison with the heart shadow on the previous examination, it was seen to be much larger. The lung fields were of satisfactory transparency. Résumé: The findings revealed the presence of a pericardial effusion.

By afternoon the dyspnea and cyanosis had become so great that the baby was placed in an oxygen chamber. The symptoms were somewhat abated, but by night the dyspnea, cyanosis, and restlessness were so great that a pericardial tap was

done. Eighty cubic centimeters of fluid were removed. The fluid was serous in character. Culture of the fluid was sterile. There was a marked improvement in dyspnea and no cyanosis for several days. The temperature remained elevated, reaching a level around 102° F. in the evening, with morning recessions. Use of the oxygen chamber was continued. Cardiac symptoms of dyspnea and cyanosis recurred, and May 25, the thirteenth day of illness, another pericardial tap was done. Ninety cubic centimeters of serous fluid were removed. Culture of this fluid was negative.

From the time of the second pericardial tap there was a gradual improvement in symptoms. Oxygen was discontinued May 27, two days after the second tap. The temperature became normal May 28, fifteen days after entrance. The white blood cell count remained high. It was 24,800 on June 9, the twenty-seventh hospital day. It still was 14,200 June 13, two days before discharge. The sedimentation rate June 9, the twenty-eighth day of illness, was 25 mm. micromethod normal 1-9; June 13, 23 mm. She was discharged June 15, the thirty-third day of illness.

The only symptoms on discharge were some dyspnea after crying and a catchy, dyspneic type of cough. She was happy, played, and was eating well. An x-ray of the heart June 9 showed that the heart shadow was decreased in width but still enlarged.

Dyspnea and cough after crying continued to exist at home for about a month. On July 12 an x-ray of the heart showed it still increased in width. The blood count was as follows: red blood cells, 4,270,000; hemoglobin, 13.3 Gm.; white blood cells, 18,500, marked shift to the left; premature forms; sedimentation rate, 14 mm. There were no longer any symptoms, however. On August 7 the white cell count was 10,200, with segmented form, 28 per cent; basophiles, 1 per cent; eosinophiles, 2 per cent; small lymphocytes, 67 per cent. The sedimentation rate was 8 mm. There was no x-ray of the heart. Clinically and by diagnostic tests the child seemed entirely well.

COMMENT

The first patient had no diagnostic laboratory or x-ray examinations; the findings and the course were that of a pericardial effusion. The diagnosis of rheumatic infection was made by clinical impression. The second patient had necessary laboratory examinations. Blood cultures were negative. Pericardial effusion fluids were sterile. Throat culture did not show any hemolytic streptococci, the usual preceding infecting organism, but cultures were not repeated. Repeated cultures will often show hemolytic streptococci where one culture does not. Neither patient had a pericardial friction rub which was detectable. Both had a fairly large effusion with no apparent involvement of the endocardium or myocardium sufficient to produce a murmur. A rheumatic pericarditis is more often part of a pancarditis with marked heart damage and murmurs.

The relatively benign course after a relatively stormy period of symptoms does not fit the picture of pyemia with pericardial involvement. In the second patient the sedimentation rate was still high a month after the temperature was normal. There is certainly more evidence in favor of these infants having had a rheumatic pericarditis than there is against it. The first patient has had no flare of rheumatic

infection nor any discoverable residual cardiac weakness. The second case is too recent to evaluate cardiac damage.

Another observation of importance is that in the second patient a pericardial tap was apparently a lifesaving measure. Pericardial taps are seldom done or need to be done and are little stressed. Almost without question this baby would not have survived without it.

SUMMARY

Pericarditis with effusion in two patients below the age of 2 years is reported as being of rheumatic etiology.

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A COMPARISON OF DIETS OF SCHOOL CHILDREN IN NEW YORK CITY IN 1917 AND 1942

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IN A WARTIME economy, nutrition of the civilian population, particularly children, is of paramount importance. This interest is reflected in the numerous published studies of the dietary habits of special groups.[1]

It is the opinion of many observers that the present generation of children is in better physical condition than a comparable group of twenty years ago. This change has been attributed to emphasis on public health education, particularly as to the consumption of protective foods.

A series of records of the daily diet of a group of New York City school children in 1917 and 1942 was made available to us for analysis. The possible effect of a wartime economy was comparable in these two years.

The data available for analysis included a group of records of every article of food consumed by 531 school children on a particular day in January, 1917, and similar records of 277 children attending the same school in December, 1942. The data were collected on a Wednesday in 1917 and on a Thursday in 1942.

The method used for obtaining the data was identical in 1917 and 1942. In the course of a penmanship lesson, the teacher asked the pupils to record the amounts of every article of food eaten the day before. There was no forewarning and no special instructions were given. Within the limits of error inherent in this type of data, the records could be assumed to be representative of the dietary habits of the school population considered.

There was no significant difference in the age constitution of the two groups. In 1917 and 1942 the ages of the children ranged from 6 to 13 years, with 80 per cent of the children between the ages of 10 and 12 years. There was an equal number of girls and boys in both groups. The neighborhood has remained representative of the white collar class in New York City, the family income ranging from $1,200 to $5,000 a year, with an average of $2,000 a year. Most of the children in both groups were born in New York of foreign-born parents.

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We wish to acknowledge our indebtedness to Dr. May G. Wilson for obtaining the records and suggesting the study.

For presentation of the average dietary consumption of various articles of food in the two periods, a random sample of 100 records of children between the ages of 10 and 12 years was selected from each group.* The per cent of children having stated portions of each article of food was calculated (Table I). A rough estimate of the adequacy of the dietary consumption in 1917 and 1942 was obtained by calculating the average number of servings of the various foods per child per day and using these factors to obtain the average protein,

TABLE I

PER CENT OF CHILDREN HAVING STATED PORTIONS OF FOOD INDICATED

ITEM	QUANTITY	PER CENT OF CHILDREN	
		1917	1942
Milk	None	53	12
	1 glass	23	19
	2 glasses	15	31
	3+ glasses	9	38
Beverages			
Coffee	1 cup	50	5
Tea	1 cup	32	3
Cocoa	1 cup	33	30
Meat	None	4	17
	1 time daily	52	51
	2 times daily	43	32
	3 times daily	1	0
Cheese	1 serving	3	17
Fish	1 serving	8	21
Eggs	None	52	55
	1 egg	23	31
	2 eggs	16	12
	3+ eggs	9	2
Fruit			
Citrus	1 serving	25	65
Other fresh	1 serving	17	21
Canned	1 serving	2	21
Stewed	1 serving	20	33
Vegetable			
Green	1 serving	32	90
Yellow	1 serving	2	30
White	1 serving	79	72
Cereal			
Cooked	1 serving	43	28
Prepared	1 serving	24	20
Bread	1 slice or more	100	97
Spaghetti, etc.	1 serving	4	18
Dessert	1 serving	49	69
Sweets	1 tsp. sugar or equivalent	64	17

fat, and carbohydrate content of the diets, the caloric intake, and the vitamin content (Table II). The calculated values were compared to the recommended daily allowances of the various food constituents proposed by the National Research Council Committee on Nutrition (1941).

*Calculations based on the random sample showed no significant difference from those carried out on the entire group.

TABLE II

CALCULATED DIETARY INTAKE AMONG SCHOOL CHILDREN (AVERAGE)

	1917	1942	RECOMMENDED ALLOWANCE*
Protein (Gm.)	78.2	79.2	70.0
Fat (Gm.)	105.8	92.6	----
Carbohydrate (Gm.)	307.4	220.6	----
Total calories	2,115	2,029	2,500
Calcium (Gm.)	0.4	0.7	1.0
Vitamin A (I.U.)	5,166	8,897	4,500
Thiamine (μg.)	896	1,167	1,200
Vitamin C (mg.)			
Total calculated	50.1	118.4	
Citrus fruit and tomato	16.4	49.7	75
Vitamin D (I.U.)	105	79	----
Riboflavin (mg.)	1.7	2.0	1.8
Niacin (mg.)	14	12	12

*Recommended Allowance for Children 10-12 Years of Age, National Research Council Committee on Nutrition, 1941.

A shortened method* was adopted for calculating the values for the dietary constituents in the 1917 and 1942 diets.[2]

A comparison of the various foods consumed in 1917 and 1942 is shown in Table I. It will be noted that the most striking changes consist in the increased consumption of milk and citrus fruit and the decreased consumption of tea and coffee. The observations in Table I are of interest.

Milk.—It is significant that in 1942 the number of children who drank milk was 88 per cent in contrast to 47 per cent in 1917. Of these, 69 per cent had two glasses or more in 1942 compared to 24 per cent in 1917. Few children drank a quart of milk in either year, but the number increased from 1 per cent in 1917 to 6 per cent in 1942.

Beverages.—The increased consumption of milk is reflected in the decreased number of children who had coffee and tea in 1942. In 1917, 50 per cent of the children had at least one cup of coffee a day in contrast to 5 per cent in 1942. The consumption of tea dropped from 32 per cent in 1917 to 3 per cent in 1942. About one-third of the children had at least one cup of cocoa a day in both years.

Meat, Fish, and Cheese.—No significant difference was observed in the overall consumption of meat and meat substitutes in 1917 and 1942. About 50 per cent of the children had meat once a day in both years. The number of children who had meat twice a day decreased by 10 per cent, and the number who had no meat increased from 4 per cent to

*Figures were obtained from "Tables of Food Values of Portions Commonly Used" by Bowes and Church.[3] These tables include appropriate corrections for loss due to cooking. All meat was considered "medium fat and medium done." For the "green" vegetables, the values for lettuce, cooked green beans, cooked spinach, cooked cabbage, cooked peas, and tomato were averaged, since these were the vegetables most frequently consumed. Cooked carrot was taken as the average "yellow" vegetable, and white boiled potato was taken as the average "white" vegetable. Since many of the children did not specify the consumption of fats and oils, one small pat of butter was included for every slice of bread indicated, and no other allowance was made for the presence of fats and oils in the diet. For calculating the other items of food, such as fish, cheese, cereal, the kind most frequently consumed was taken as the average. For obtaining the average dietary intake, the average number of servings per child per day of each item was multiplied by the appropriate figures in the Bowes and Church tables, giving the average calculated intake of the various dietary constituents.

17 per cent. Consideration must be given to the possible difficulties encountered in purchasing meat and the rise in prices in December, 1942, which appears to be reflected in the increased number of children who had fish and cheese. The number of children who had at least one serving of either fish or cheese increased from under 10 per cent in 1917 to about 20 per cent in 1942.

Eggs.—No change was observed in the number of children who ate eggs on the day that the data were collected. In both years eggs were not listed in about 50 per cent of the records. Of those having eggs, there was a small decrease in the number of children who had more than one egg in 1942.

Fruit.—The increase in the consumption of citrus fruit is noteworthy. In 1942, 65 per cent of the children had at least one serving of orange, grapefruit, or tangerine in contrast to 25 per cent in 1917. This may be attributed to the increased emphasis laid on the consumption of citrus fruit in the past few years as well as to its increased availability.

In both years about 20 per cent of the children had at least one serving of fresh fruit other than citrus fruit, usually apple. The consumption of canned fruit, such as pears, peaches, and pineapple, has increased from 2 per cent in 1917 to 21 per cent in 1942, probably reflecting the wider use of canned fruits in recent years. The consumption of stewed fruits, particularly prunes and applesauce, has increased from 20 per cent in 1917 to 33 per cent in 1942.

Vegetables.—The most striking difference in the consumption of vegetables has been the increase in the quantity and variety of the "green" vegetables consumed in 1942, particularly lettuce, and tomato. In 1942, 90 per cent of the children had at least one serving of "green" vegetable a day in contrast to 32 per cent in 1917. Of these, 59 per cent had two servings of green vegetable in 1942, and only 3 per cent of the children in 1917 had more than one serving. Of the cooked green vegetables, peas, beans, cabbage, and spinach were the most frequently consumed in both years.

Carrot was the "yellow" vegetable most frequently consumed in both 1917 and 1942. The number of children having one serving of "yellow" vegetable had increased from 2 per cent in 1917 to 30 per cent in 1942.

Potato was eaten at least once a day by about 75 per cent of the children in 1917 and 1942. Of these, about 25 per cent had potato twice a day in both years. It must be noted, however, that potato was frequently the only vegetable which appeared in the 1917 diet.

Cereal.—Cooked cereal has shown a decrease from 43 per cent in 1917 to 28 per cent in 1942. About 20 per cent of the children in both years had prepared cereal. The variety of cereals in the diet, both prepared and cooked, is much greater today than in 1917, although oatmeal and corn flakes remain the most frequently consumed.

Bread and Other Grain Products.—The overall consumption of bread has shown a decrease since 1917. In 1942, 3 per cent of the children ate no bread, while in 1917, every child had at least one slice. The average number of slices of bread consumed per day has decreased from six slices in 1917 to four slices in 1942. It was also noted that many of the children in the 1917 group ate nine and ten slices of bread a day, which was a rare occurrence in 1942. There was no indication in the data as to whether the bread consumed was made of white or whole grain flour.

The per cent of children who had at least one serving of spaghetti or macaroni increased from 4 per cent in 1917 to 18 per cent in 1942. The proportion of children of Italian descent had not changed.

Dessert and Sweets.—The number of children who had at least one serving a day of cake, pie, or pudding increased from about 50 per cent in 1917 to 70 per cent in 1942.

A decrease was observed in the use of sugar, jams, jellies, and candy. Those having at least one spoonful of sugar, or its equivalent, decreased from 64 per cent in 1917 to 17 per cent in 1942.

In Table II the calculated average diets in 1917 and 1942 are summarized for comparison with the Recommended Daily Allowances of the Committee on Food and Nutrition of the National Research Council.

The caloric intake in both years appears to be lower than the recommended allowance. This may be attributed in large part to the method of averaging used in the analysis. The intake of protein was apparently adequate in both years, and the high value for carbohydrate, particularly in 1917, is noteworthy.

The calculated calcium intake, using milk, cocoa, and cheese as the chief sources, has increased from 0.4 Gm. in 1917 to 0.7 Gm. in 1942. Since few children in either year drank a quart of milk a day, it is probable that the recommended allowance of 1.0 Gm. of calcium a day was attained by only 9 per cent of the children in 1917 and 38 per cent in 1942.

In general, the vitamin content of the 1942 diet appeared to be more adequate than that of the 1917 diet. The average vitamin A content was greater than the recommended allowance in both 1917 and 1942. The fact that the 1942 diet contained twice as much calculated vitamin A as the recommended allowance may be attributed to the high consumption of green leafy vegetables, particularly lettuce and spinach. The intake of thiamine was low in 1917 and almost adequate in 1942. There was probably a greater loss of thiamine due to cooking methods in 1917 than in 1942, but this was not accounted for in the calculations. The vitamin D content could not be evaluated, and the values for riboflavin and niacin appear to be within normal limits.

The vitamin C content of the diet was probably inadequate in both years. In 1917, 16 mg. were obtained from citrus fruit and tomato. In 1942, 50 mg. were similarly obtained. For all other sources of vitamin C in the diet, the loss due to cooking is appreciable. It has been esti-

mated that not more than 10 mg. of vitamin C are available from these sources.[4] This amount is insufficient to raise the amount of vitamin C in the average diets of 1917 and 1942 to within the recommended allowance.

It is probable that present cooking methods are far superior to those used in 1917. For this reason, the values for the water-soluble vitamins which were calculated for 1917 are probably high.

COMMENT

Within the limits inherent in dietary histories, the following conclusions may be made:

There is a definite improvement in the dietary habits of the present generation in the school population studied. This is due in large measure to the influence of public health education. The increased consumption of milk, citrus fruits, and vegetables is noteworthy. The greater availability of fresh fruits and vegetables due to the extension of facilities for their transportation to the city is also a contributory factor.

It cannot be concluded that in 1942 the dietary histories reflected an optimum daily diet. The consumption of protective foods, particularly foods containing vitamin C, is probably not adequate. Further emphasis is necessary, particularly as to the comparative nutritive values of different foods and the preferred methods for preparing cooked foods.

The majority of the children in both periods had inadequate breakfasts, and frequently an inadequate lunch. The need for school lunches, even for children who are not considered to be underprivileged, is apparent.

Data were not available for comparing the nutritional status of children in 1917 and 1942. Children attending special open air or nutrition classes were not included in this survey in either year.

The requirements of food rationing at the present time afford public health authorities an unusual opportunity to consolidate the gains already made and to influence the improvement of the dietary habits of school children.

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SEASONAL VARIATIONS IN WEIGHT, HEIGHT, AND APPEARANCE OF OSSIFICATION CENTERS

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THE present study is an investigation of seasonal differences in weight and height gain, and, in one aspect, of skeletal development, in 133 children during the age span from 12 to 60 months.

A number of studies indicating seasonal influences on weight and height have been made, particularly by European workers. Saunders[1] has discussed the evidence from these sources. However, in American research the evidence for seasonal variations, particularly in height, is somewhat conflicting. Of ten American studies summarized by Marshall,[2] eight indicated a seasonal variation in weight. Of the six studies which also included observation on height, four found no differences which could be attributed to seasonal influences.

No studies, to our knowledge, have been reported as yet on possible seasonal variations in the appearance of ossification centers in the body.

MATERIAL AND METHODS

The children used in the present study, sixty-five boys and sixty-eight girls from southwestern Ohio, were selected from the Fels Research Institute on the basis of completeness of records from 1 through 5 years of age. These children were measured and roentgenograms were taken at birthdays and half-birthdays. Weight was taken without clothes, height represents horizontal body length. Increments of weight and height for each six months of growth have been calculated and means and variabilities determined in an earlier publication.[3]

Complete series of roentgenograms, taken at each visit, made it possible to determine which of the selected centers of ossification had appeared during each interval. The centers considered have been discussed and norms for time of appearance presented in previous studies.[4, 5] The sexes have been treated both separately and in combination.

Table I presents mean half-yearly and yearly increments of weight and height, together with mean number of ossification centers present, by month of birth and for the total group.

The means shown in Table I represent the age span 12 through 60 months. They are thus derived, for each child, from four increments representing the first half-years of growth during the second, third, fourth, and fifth years of life and from four increments representing the second half-years of growth during this period.

Since the children were measured at birthdays and half-birthdays, certain seasonal periods of growth coincide. Thus, the gain during the

From The Samuel S. Fels Research Institute, Antioch College.

TABLE I

MEAN HALF YEARLY AND YEARLY INCREMENTS IN WEIGHT, HEIGHT, AND NUMBER OF NEW OSSIFICATION CENTERS BY MONTH OF BIRTH*

MONTH OF BIRTH	WEIGHT (KG.)				HEIGHT (CM.)				OSSIFICATION (NUMBER OF CENTERS)			
	NUMBER OF ITEMS	MEAN INCREMENT			NUMBER OF ITEMS	MEAN INCREMENT			NUMBER OF ITEMS	MEAN NUMBER		
		FIRST HALF OF YEAR	SECOND HALF OF YEAR	YEAR		FIRST HALF OF YEAR	SECOND HALF OF YEAR	YEAR		FIRST HALF OF YEAR	SECOND HALF OF YEAR	YEAR
Boys												
Jan.	8	1.14	1.26	2.40	8	5.2	4.0	9.2	7	4.3	4.3	8.6
Feb.	18	.89	1.31	2.20	18	4.8	4.1	8.9	20	6.4	3.8	10.2
March	23	.98	1.12	2.10	23	4.9	3.9	8.8	18	4.6	5.7	10.3
April	15	1.23	1.01	2.24	15	4.6	4.0	8.6	15	5.7	5.1	10.8
May	38	1.24	1.07	2.31	37	4.8	4.5	9.3	33	4.5	4.1	8.6
June	24	1.46	1.10	2.56	24	5.1	4.2	9.3	29	5.4	5.1	10.5
July	14	1.02	.80	1.82	15	4.4	4.3	8.7	15	5.3	4.9	10.2
Aug.	11	1.34	.61	1.95	11	4.6	4.2	8.8	9	4.9	6.3	11.2
Sept.	4	1.22	.72	1.94	4	4.3	5.2	9.5	6	5.0	6.5	11.5
Oct.	18	1.32	1.11	2.43	19	4.7	4.6	9.3	18	5.1	4.5	9.6
Nov.	7	1.10	.93	2.03	7	4.1	4.3	8.4	7	6.9	4.0	10.9
Dec.	38	1.19	1.14	2.33	38	4.9	3.7	8.6	40	4.8	4.4	9.2
Mean	(218)	1.19	1.06	2.25	(219)	4.8	4.2	9.0	(217)	5.2	4.7	9.9
Girls												
Jan.	27	.93	1.08	2.01	27	5.0	3.9	8.9	25	5.1	3.1	8.2
Feb.	16	.98	1.10	2.08	16	5.0	4.2	9.2	20	6.5	3.3	9.8
March	19	.64	1.30	1.94	19	4.8	3.4	8.2	16	4.9	3.4	8.3
April	21	1.07	1.01	2.08	22	4.6	4.2	8.8	17	5.3	3.2	8.5
May	15	1.21	1.15	2.36	15	4.9	4.6	9.5	15	5.1	4.5	9.6
June	18	1.20	1.08	2.28	17	4.7	4.5	9.2	14	4.1	4.2	8.3
July	25	1.29	.95	2.24	25	4.6	4.3	8.9	24	5.1	3.5	8.6
Aug.	18	1.62	.96	2.58	18	4.6	4.9	9.5	19	4.2	3.7	7.9
Sept.	15	1.63	.83	2.46	14	4.8	4.7	9.5	14	4.6	4.4	9.0
Oct.	17	1.14	1.10	2.24	18	4.3	5.0	9.3	17	5.8	4.5	10.3
Nov.	28	.89	.90	1.79	28	4.5	4.2	8.7	29	5.9	3.9	9.8
Dec.	22	.94	1.21	2.15	21	5.1	4.0	9.1	20	5.9	3.3	9.2
Mean	(241)	1.10	1.05	2.15	(240)	4.7	4.3	9.0	(230)	5.3	3.7	9.0

*Derived from the four years of growth between the ages of 1 and 5 years.

first half of a year for a child born in January covers the same six months—January to July—as the gain for the second half of a year for a child born in July. January and July children may, therefore, be combined, since, being measured semiannually, their seasonal periods of growth have the same end points. The two values obtained represent weighted means for growth from January to July and from July to January. Each mean contains the same number of items. Other groups may be combined in the same fashion as follows: children born in February with children born in August, those born in March with those in September, those in April with those in October, those in May with those in November, and those in June with those in December.

Within each of these six paired groups it is possible to compare the growth during a certain six months of the year with the growth during the other six months of the year. Since each of these paired groups is

one month removed in time of measurement from its adjoining group, it is possible to make six comparisons of differences in growth between six-month periods, the comparisons moving in one-month intervals around the calendar.

These six comparisons of growth differences are independent estimations of possible seasonal variations in that the values which go to make each set are not contained in any of the other five sets.

Since the method of analysis is the same for each of the three items being considered, further description will be given in the following section.

RESULTS

Weight.—Table II shows six independent semiannual comparisons of weight gain for each sex and for the sexes combined.

These comparisons represent the difference between weight gain for six months of the year and weight gain for the other six months.* For example, in boys measured in February and August, the weight gain from August to February was 64 per cent of the mean yearly weight gain, while the weight gain from February to August was 36 per cent of the mean yearly weight gain for these boys. On the other hand, in boys measured in October and April, the weight gain for each of the two half-year periods was the same.

These seasonal differences in weight gain, based on six independent sets of values for each sex, are shown graphically in Fig. 1.

In boys the six months of maximum weight gain was from August to February; the six months of minimum weight gain, from February to August. In girls the six months of maximum weight gain was from September to March; the six months of minimum weight gain, from March to September. In both sexes the weight gain from April to October was equal to the weight gain from October to April, and the weight gain for boys from May to November was equal to the weight gain from November to May.

Height.—Table III shows six independent semiannual comparisons of height gain for each sex and for the sexes combined.

In boys the six months of maximum height gain was from March to September; the six months of minimum height gain, from September to March. The same pattern held for the girls. The height gain for girls from May to November was equal to the height gain from November to May. This same period shows a low difference in height gain for the boys, who are also low in the April to October and the February to August series.

*As shown in Table I, there is some difference, within each category, between the mean of the first half-year increments and the mean of the second half-year increments. This was compensated for by applying a correction factor to individual values so that the means were equal to each other and their sums equal to the yearly mean. These factors were as follows: boys' weight, .9454 times the first half-year values and 1.0613 times the second half-year values; girls' weight, .9773 and 1.0238; boys' height, .9375 and 1.0714; girls' height, .9574 and 1.0465; boys' ossification, .9519 and 1.0532; and girls' ossification, .8491 and 1.2162. These corrections removed that part of the difference between growth gains which was not seasonal. It was unnecessary to correct for differences due to month of birth, per se, since the analysis was done by percentages of yearly gain in every instance.

TABLE II

SEASONAL DIFFERENCES IN WEIGHT GAIN AS SHOWN BY SIX INDEPENDENT COMPARISONS

BOYS				GIRLS				BOYS AND GIRLS COMBINED			
PERIOD OF GROWTH	NUMBER	GAIN (KG.)	PER CENT OF YEARLY GAIN	PERIOD OF GROWTH	NUMBER	GAIN (KG.)	PER CENT OF YEARLY GAIN	PERIOD OF GROWTH	NUMBER	GAIN (KG.)	PER CENT OF YEARLY GAIN
July to Jan.	22	1.10	54	July to Jan.	52	1.18	56	July to Jan.	74	1.14	55
Jan. to July	22	.93	46	Jan. to July	52	.94	44	Jan. to July	74	.94	45
Aug. to Feb.	29	1.34	64	Aug. to Feb.	34	1.37	59	Aug. to Feb.	63	1.36	61
Feb. to Aug.	29	.77	36	Feb. to Aug.	34	.97	41	Feb. to Aug.	63	.87	39
Sept. to March	27	1.18	58	Sept. to March	34	1.44	69	Sept. to March	61	1.31	64
March to Sept.	27	.84	42	March to Sept.	34	.65	31	March to Sept.	61	.74	36
Oct. to April	33	1.17	50	Oct. to April	38	1.07	50	Oct. to April	71	1.12	50
April to Oct.	33	1.17	50	April to Oct.	38	1.09	50	April to Oct.	71	1.13	50
Nov. to May	45	1.12	50	Nov. to May	43	.98	49	Nov. to May	88	1.05	50
May to Nov.	45	1.13	50	May to Nov.	43	1.01	51	May to Nov.	88	1.07	50
Dec. to June	62	1.15	47	Dec. to June	40	1.01	45	Dec. to June	102	1.08	46
June to Dec.	62	1.28	53	June to Dec.	40	1.21	55	June to Dec.	102	1.24	54

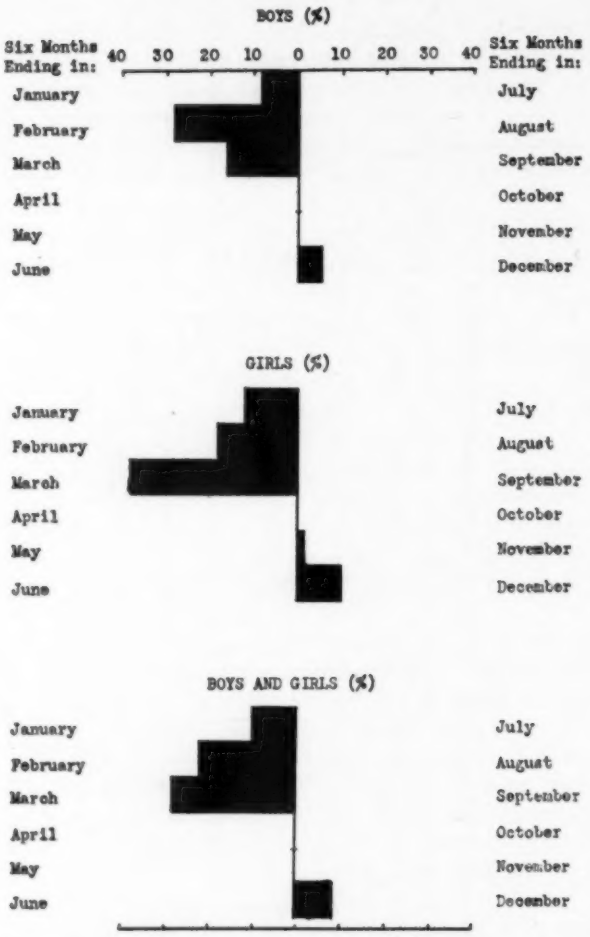

Fig. 1.—Seasonal differences in weight gain. Six semiannual comparisons for each sex and for the sexes combined based on measurements taken at the months indicated. Excess of gain in weight in one six-month period over gain in weight in the other six-month period is shown in percentage of yearly gain.

These differences are shown graphically in Fig. 2.

Appearance of Ossification Centers.—Table IV shows six independent semiannual comparisons of the number of ossification centers appearing for each sex and for the sexes combined.

In boys the six-month period during which new ossification centers appeared most rapidly was from February to August, the six months of minimum advance being from August to February. In girls the same pattern held, with the addition of an equally large increase in December to June over June to December.

These differences are shown graphically in Fig. 3.

TABLE III

SEASONAL DIFFERENCES IN HEIGHT GAIN AS SHOWN BY SIX INDEPENDENT COMPARISONS

BOYS				GIRLS				BOYS AND GIRLS COMBINED			
PERIOD OF GROWTH	NUMBER	GAIN (CM.)	PER CENT OF YEARLY GAIN	PERIOD OF GROWTH	NUMBER	GAIN (CM.)	PER CENT OF YEARLY GAIN	PERIOD OF GROWTH	NUMBER	GAIN (CM.)	PER CENT OF YEARLY GAIN
July to Jan.	23	4.2	47	July to Jan.	52	4.2	47	July to Jan.	75	4.2	47
Jan. to July	23	4.7	53	Jan. to July	52	4.7	53	Jan. to July	75	4.7	53
Aug. to Feb.	29	4.4	49	Aug. to Feb.	34	4.4	47	Aug. to Feb.	63	4.4	48
Feb. to Aug.	29	4.5	51	Feb. to Aug.	34	5.0	53	Feb. to Aug.	63	4.8	52
Sept. to March	27	4.1	46	Sept. to March	33	4.0	45	Sept. to March	60	4.1	46
March to Sept.	27	4.7	54	March to Sept.	33	4.7	55	March to Sept.	60	4.7	54
Oct. to April	34	4.4	49	Oct. to April	40	4.3	47	Oct. to April	74	4.4	48
April to Oct.	34	4.6	51	April to Oct.	40	4.8	53	April to Oct.	74	4.7	52
Nov. to May	44	4.6	51	Nov. to May	43	4.5	50	Nov. to May	87	4.6	51
May to Nov.	44	4.5	49	May to Nov.	43	4.5	50	May to Nov.	87	4.5	49
Dec. to June	62	4.6	52	Dec. to June	38	4.8	53	Dec. to June	100	4.7	52
June to Dec.	62	4.3	48	June to Dec.	38	4.3	47	June to Dec.	100	4.3	48

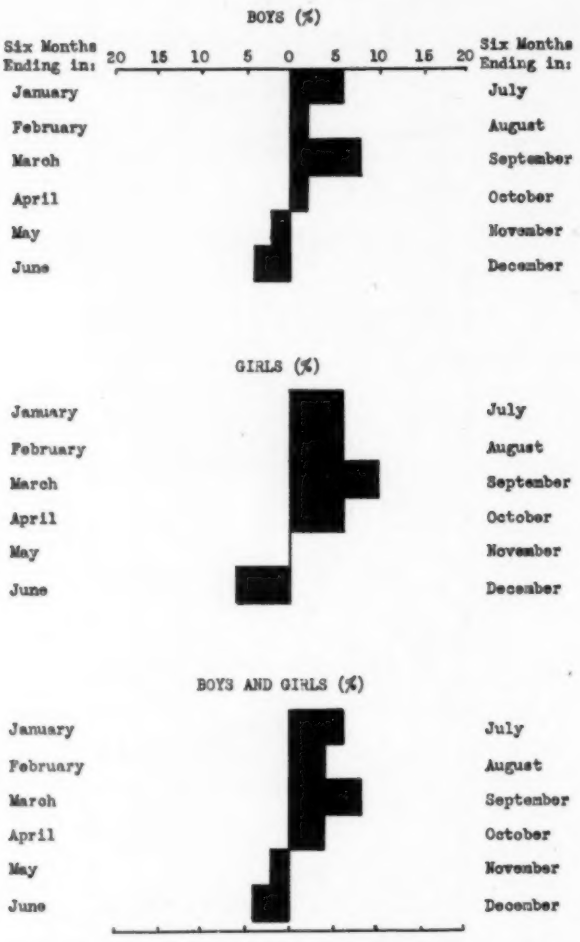

Fig. 2.—Seasonal differences in height gain. Six semiannual comparisons for each sex and for the sexes combined based on measurements taken at the months indicated. Excess of gain in height in one six-month period over gain in height in the other six-month period is shown in percentage of yearly gain. Note that the pattern of seasonal differences is opposite to Fig. 1, *Weight*.

DISCUSSION

Fig. 4 presents the six independent semiannual comparisons for each of the three items under consideration, the values for the boys and girls being combined.

Three facts are apparent from an examination of Fig. 4. First, in each of the three items there appears to be a clear-cut pattern of differences between semiannual comparisons. This pattern may be

TABLE IV

SEASONAL DIFFERENCES IN APPEARANCE OF OSSIFICATION CENTERS AS SHOWN BY SIX INDEPENDENT COMPARISONS

BOYS				GIRLS				BOYS AND GIRLS COMBINED (NUMBER OF CENTERS)			
PERIOD OF GROWTH	NUMBER	GAIN	PER CENT OF YEARLY GAIN	PERIOD OF GROWTH	NUMBER	GAIN	PER CENT OF YEARLY GAIN	PERIOD OF GROWTH	NUMBER	GAIN	PER CENT OF YEARLY GAIN
July to Jan.	22	4.8	49	July to Jan.	49	4.0	48	July to Jan.	71	4.4	49
Jan. to July	22	4.9	51	Jan. to July	49	4.3	52	Jan. to July	71	4.6	51
Aug. to Feb.	29	4.2	40	Aug. to Feb.	39	3.8	43	Aug. to Feb.	68	4.0	42
Feb. to Aug.	29	6.3	60	Feb. to Aug.	39	5.0	57	Feb. to Aug.	68	5.6	58
Sept. to March	24	5.7	53	Sept. to March	30	4.0	45	Sept. to March	54	4.8	49
March to Sept.	24	5.0	47	March to Sept.	30	4.8	55	March to Sept.	54	4.9	51
Oct. to April	33	5.1	50	Oct. to April	34	4.4	47	Oct. to April	67	4.8	49
April to Oct.	33	5.0	50	April to Oct.	34	5.0	53	April to Oct.	67	5.0	51
Nov. to May	40	4.7	52	Nov. to May	44	5.2	53	Nov. to May	84	5.0	53
May to Nov.	40	4.3	48	May to Nov.	44	4.6	47	May to Nov.	84	4.4	47
Dec. to June	69	4.9	51	Dec. to June	34	5.0	57	Dec. to June	103	5.0	54
June to Dec.	69	4.8	49	June to Dec.	34	3.8	43	June to Dec.	103	4.3	46

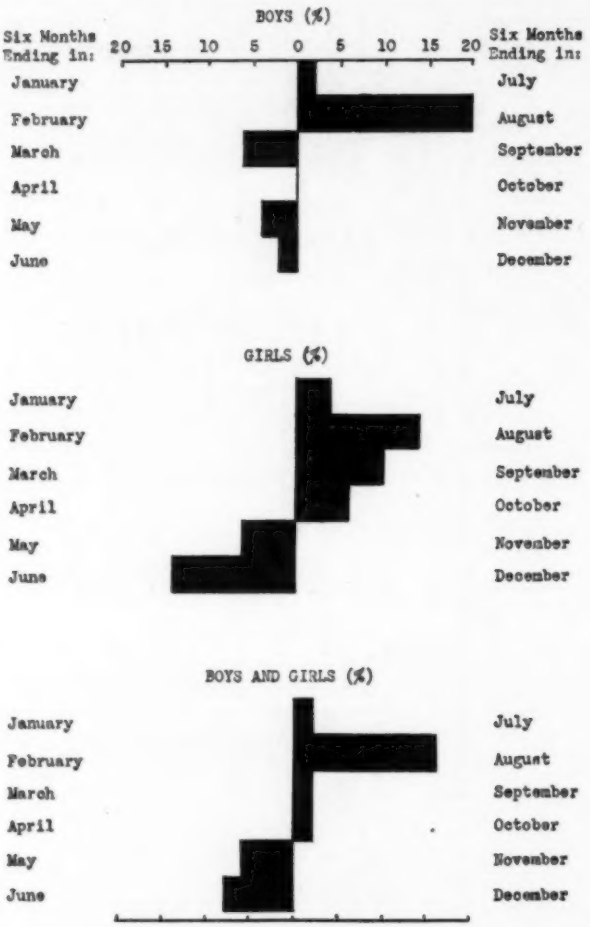

Fig. 3.—Seasonal differences in appearance of ossification centers. Six semiannual comparisons for each sex and for the sexes combined based on observations made at the months indicated. Excess of number of centers appearing in one six-month period over number of centers appearing in the other six-month period is shown in percentage of centers appearing during that year.

ascribed to "seasonal" influences without at this time attempting to say just what causal factor or factors motivate these seasonal changes.*

Second, the pattern for height and the pattern for appearance of ossification centers appear to be essentially parallel. The peak for height increase comes in the six months between March and September; the peak for the appearance of ossification centers comes in the six months between February and August. Without exception, the

*A subsequent paper will make this attempt, incorporating growth data on the first year of life and investigating seasonal differences in illness, nutrition, climate, and other factors in these same children.

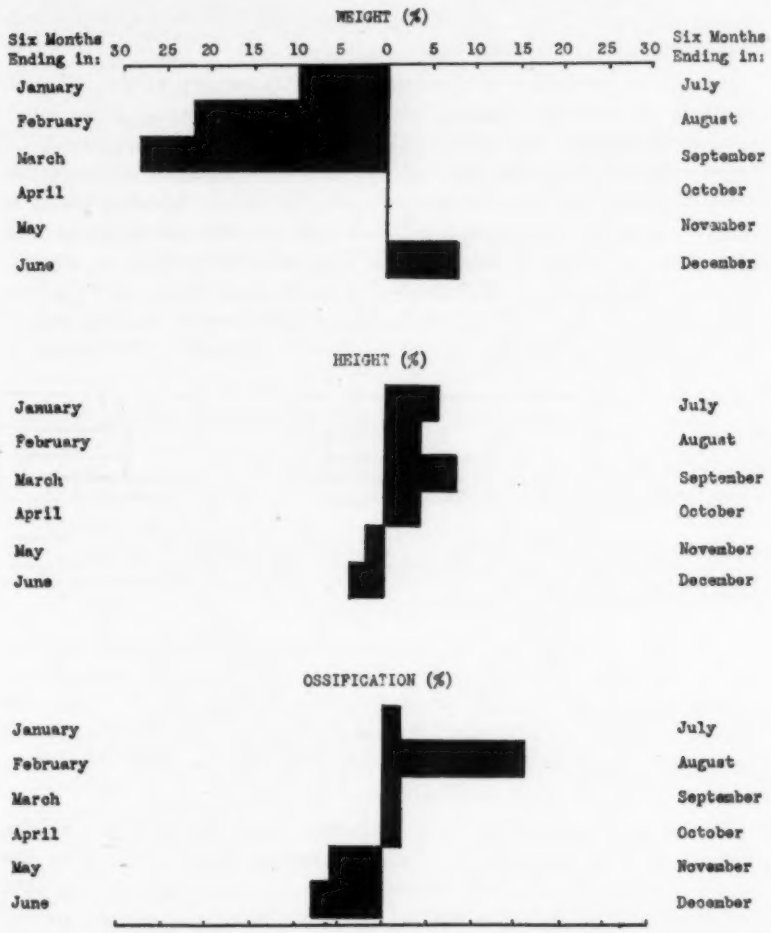

Fig. 4.—Seasonal differences in weight and height gain and in appearance of ossification centers, boys and girls combined. Excess of gain in one six-month period over gain in the other six-month period is shown in percentage of yearly gain.

six-month periods of greater gain in height, in each of the comparisons, correspond to the same period of greater gain in number of ossification centers appearing.

Third, the seasonal pattern for weight is opposite to the seasonal patterns for the other two items. Since growth in height is primarily an expression of growth of the skeleton, this means that weight gain and skeletal growth react in opposite fashion during the same season of the year. This does not necessarily mean that the factor or factors causing an acceleration in weight gain are the same factors that cause a deceleration in skeletal growth and ossification and vice versa. This may

or may not be the case. However, it is evident that an opposite effect is taking place at the same time.

Because of the nature of the data used in the present study, it is not justifiable to construct seasonal growth curves representing a continual pattern throughout the year. Although measurements are taken in every month of the year for each sex, measurements are made on an individual child not every month, but semiannually. Nevertheless, it is entirely possible, by examining the paired comparisons given in the preceding paragraphs, to determine seasonal differentials in growth within closer limits than six months. Such an analysis is shown in Fig. 5, wherein, for each item the maximum and minimum periods of weight and height gain and appearance of ossification centers are indicated.

Item	Jan.	Feb.	Mch.	Apr.	May	June	July	Aug.	Sep.	Oct.	Nov.	Dec.
Weight					VALLEY						PEAK	
Height					PEAK						VALLEY	
Ossification				PEAK						VALLEY		

Fig. 5.—Comparison of seasonal cycles in weight and height gain and in appearance of ossification centers. Constructed by analysis of degree of monthly representation in each of the six semiannual comparisons of seasonal differences. *Peak* represents the time of the year of fastest gain in weight or height or in ossification; *valley* represents the period of slowest gain.

The peak of weight gain is between October and December and the valley, between April and June. This picture is exactly reversed for height gain, with the peak between April and June and the valley between October and December. The ossification cycle parallels the height but is one month in advance, the peak lying between March and May and the valley, between September and November.

The seasonal differences just indicated are pronounced for weight, moderate for ossification, and slight for height. Their significance lies in the consistently parallel patterns which the sexes show within each category, as well as in the gradual shifts in differences between seasons as the comparisons shift in monthly steps. In only one instance in the eighteen comparisons between sexes—ossification in the March to September category—do the sexes fail to agree.

If the fluctuations in individual curves of growth are inspected, it becomes apparent that these fluctuations, for example, in weight, are responsive to seasonal influences. When semiannual measurements are so taken that one-half-year of growth contains the season of maximum weight gain and the other half-year of growth contains the season of minimum weight gain, this phenomenon becomes most evident. During the half-year of maximum seasonal weight gain, the fluctuations are predominately upward, in the direction of acceleration. During the half-year of minimum seasonal weight gain, the fluctuations of individual growth curves are predominately downward.

If, on the other hand, semiannual measurements are so taken that seasonal gains during each half of the year are equal, fluctuations of individual growth curves in weight tend to be distributed evenly upward and downward.

Fluctuations in individual weight curves, therefore, are, in part at least, ascribable to seasonal influences, and in curves plotted on the basis of semiannual measurements the pattern of fluctuations is determined in large measure by the months in which these semiannual measurements are taken.

In view of our findings and those of others a word of caution with regard to analyses of individual growth fluctuations seems in order. Particularly when deviations from restricted paths of individual growth patterns are accepted as indications of inadequate physical status, it would be well to take seasonal factors into account when examining such deviations.

SUMMARY AND CONCLUSIONS

1. In the present study we investigated seasonal variations in weight and height and in the appearance of specific ossification centers in 133 children during the age span from 12 to 60 months.

2. Seasonal variations, similar in each sex, were found to exist in each of the three categories.

3. The variation in weight was pronounced; in ossification, moderate; and in height, slight.

4. Seasonal variations in height and ossification were parallel and opposite to the seasonal variation in weight.

5. The period of maximum weight gain was from October to December; minimum weight gain, from April to June.

6. The period of maximum height gain was from April to June; minimum height gain, from October to December.

7. The period of maximum rate of appearance of ossification centers was from March to May; minimum rate of appearance, from September to November.

8. Fluctuations in individual growth curves, based on semiannual measurements, were shown to be sharply responsive to seasonal differences in rate of growth.

9. Analyses of deviations in individual growth curves should take into consideration the season of the year which is covered by the interval between measurements.

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ACUTE ATAXIA OF UNKNOWN ORIGIN IN CHILDREN

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THE purpose of this report is to call attention to an acute nervous system disturbance manifesting chiefly ataxic symptoms. Although similar instances have been noted in the past,[1-6] they have been distinctly rare. In the great epidemic of encephalitis following the influenza epidemic of 1917 and 1918, only rare instances of purely ataxic disturbances were noted.[7] Cases with similar symptoms, but of a progressive and chronic type, have been recorded under the term dyssynergia cerebellaris progressiva and dyssynergia cerebellaris myoclonica by Hunt,[8, 9] who, in 1914 and 1920, described this condition as a primary system disease of the cerebellum and its efferent pathways, the dentate system. The causative factor was thought to be a primary atrophic process, an interpretation borne out by pathologic studies in one case.

Whether we should ascribe the name encephalitis to the condition which we are presenting or merely call it an acute nonsuppurative disturbance of the nervous system is debatable. Our group of cases is without the benefit of pathologic findings and without a known etiologic factor. The history and course of the disturbances do, however, suggest an acute infectious or postinfectious or related process in some of the cases.

The following seven cases were observed in the Babies Hospital in the course of three years' time. The neurologic signs are summarized in Table I, the laboratory findings in Table II. Abstracts of the patients' records are presented in the order of severity of their symptoms, with a summary of the cases in Table III.

CASE 1.—A. Q., 4½ years of age, had a transient temperature of 104° F. and a sore throat nine days before entry. These symptoms kept her out of school for only one day. Three days before admission she became drowsy and her temperature rose to 102° F. She soon complained of malaise and refused to sit up or to raise her head from the pillow. She vomited several times. It was then noted that her hands shook whenever she attempted to reach for an object and that when she did attempt to sit up, there was marked unsteadiness of the head and body. She complained of slight headache.

She did not appear to be severely ill. There was a slight tendency to internal deviation of the eyes. Nystagmus was not noted and meningeal signs were absent. The deep reflexes were equal and somewhat hyperactive. The abdominal reflexes were present and equal. There was no focal motor weakness. She had a marked intention tremor. The finger-to-nose and the heel-to-knee tests were poorly performed. When she sat up there was a marked ataxia with wavering of the trunk

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TABLE I

NEUROLOGIC SIGNS IN SEVEN CASES OF ACUTE ATAXIA OF UNKNOWN ORIGIN IN CHILDREN

CASE	NEUROLOGIC SIGNS
1 (A. Q.)	Ataxic gait Falling in Romberg position Inability to walk because of extreme ataxia Intention tremor Tremor of extremities, head, neck, and trunk
2 (J. O.)	Ataxic gait Falling in Romberg position Ataxia in extremities
3 (S. L.)	Ataxic gait Falling in Romberg position Intention tremor Slurred speech Nystagmus Tremor of head, neck, and trunk, while walking or sitting Myoclonic movements
4 (M. M.)	Ataxic gait Falling in Romberg position Intention tremor Nystagmus Tremor of all extremities, head, and trunk Slurred speech
5 (F. M.)	Ataxic gait Intention tremor Myoclonic movements
6 (J. S.)	Ataxic gait Intention tremor Tremor of head, arms, and trunk while sitting or standing Nystagmus
7 (G. S.)	Ataxic gait Intention tremor of all extremities Tremor of head and trunk while sitting or standing Nystagmus

from side to side. The neck was then held stiffly in an attempt to steady the head. She could not be induced to stand because of her fear of falling and her unsteadiness in walking.

She remained in the hospital for five days. By the third day there was marked improvement and by the time of discharge there were no abnormal neurologic signs. At no time was there any unnatural change in her mental condition.

CASE 2.—J. O., 3 years of age. Four days before entry, without any previous illness, the parents noticed that the patient was unsteady on his legs when arising in the morning. He refused his breakfast and was thought to be pale. During the day he fell down several times when he attempted to walk. He improved toward evening, but the same episodes were repeated on subsequent days. He vomited twice during these three days.

On admission he did not appear to be ill. The extraocular movements were normal, as were the eye grounds. The muscular strength appeared to be good. The reflexes were present, active and equal. He refused to perform the finer tests for ataxia. When persuaded to walk he did so on a wide base and tended to fall, although in no consistent direction.

The child remained in the hospital for three weeks. During the first week there was little change in his condition, the ataxia in walking and incoordination in the

extremities persisting, but during the subsequent period he showed gradual improvement. At the time of discharge he appeared to be quite normal. There were no obvious mental changes. He was followed in the Vanderbilt Clinic and at the end of two months appeared to be perfectly normal.

CASE 3.—S. L., 2 years of age. Four days before entry, without any history of preceding disease, the parents noticed that the patient's gait had become wobbly. It was thought that he tended to deviate toward the left side. There were no other complaints. He had not vomited or had headaches.

On admission he did not appear to be ill. He was able to move all his extremities well but did so with a coarse intention tremor. When he attempted to walk, he did so on a wide base and had a tendency to fall, although in no consistent direction. There was a transient nystagmus, but as a rule the extraocular movements were well performed. The eye grounds were normal. The reflexes were present, active and equal. There were no meningeal signs. Two days after entry he became much worse. There developed tremors of all the extremities and of the neck, and there were many irregular extraocular movements and choreic and myoclonic movements. His ataxia progressed until he was unable to walk or to stand. There was an extreme degree of hypotonia of all the musculature. At one time there was thought to be a right facial weakness. All these manifestations became worse whenever the patient was disturbed. He had periods of marked irritability. He was transferred to the Neurological Institute, where he remained for six weeks. During the end of this period he began to show signs of improvement, although he was still unable to walk at the time of discharge.

After discharge the improvement continued until the neurologic signs had almost disappeared. Four months after discharge he was readmitted to the Babies Hospital with an abdominal mass. At this time there was no suggestion of his previous troubles. The mass was found to be an undifferentiated malignant tumor that was inoperable and caused death after about two months. Autopsy findings showed a sympathicoblastoma of the left adrenal gland. The brain was not examined since permission could not be obtained for a head examination. The spinal cord, however, showed no abnormalities. It hardly seemed possible that the previously existing condition could have been due to the final illness, since he had recovered almost completely before his fatal illness appeared. However, in all cerebellar lesions there may be good compensation for the cerebellar defect, and we must include an element of doubt as to possible metastatic involvement of the cerebellum.

CASE 4.—M. M., 3½ years of age. Six weeks before entry this patient had a mild upper respiratory infection. Two weeks later she became more irritable than usual and began to have temper tantrums. Two weeks before entry she began to have a trembling of the body which was almost constantly present. She became clumsy in her movements and spoke indistinctly. Three days before entry she began to vomit everything that she took by mouth.

She did not appear to be severely ill. She had involuntary jerking movements of the arms and legs when active, but they subsided when at rest. The eyes showed a nystagmus. The fundi were normal. Her speech was markedly slurred. The deep reflexes were present and equal and were thought to be hyperactive. There was a distinct tremor in the finger-to-nose and the heel-to-knee tests. On walking, marked ataxia was present. The Romberg test was positive, although she did not tend to fall in any consistent direction. There was coarse, irregular tremor of the head, neck, trunk, and lower extremities in sitting, standing, or walking.

During the first three weeks in the hospital the picture changed but little. Her speech became more distinct. After the third week there began a gradual subsidence of ataxic symptoms. At the time of discharge she was greatly improved.

TABLE II

LABORATORY FINDINGS

CASE	SPINAL FLUID					BLOOD (WASSERMANN OR KLINE)	TUBERCULIN	X-RAY OF SKULL	PNEUMOENCEPHALOGRAM
	CELLS PER C.MM.	PROTEIN (MG. PER 100 C.C.)	SUGAR (MG. PER 100 C.C.)	WASSERMANN	GOLD CURVE				
1	6	16	53	Negative	Normal	Negative		Negative	
2	16	38.6	62	Negative	Normal	Negative	Negative	Negative	
3	8	Pandy –	Present	Negative	Normal	Negative	Negative	Negative	
4	1	Pandy –	62	Negative	Normal	Negative	Negative	Negative	
5	3	13	49	Negative	Normal	Negative	Negative	Negative	Moderate atrophy
6	20	20	73	Negative	Normal	Negative	Negative	Negative	Moderate atrophy
7	4	Pandy –	Present	Negative	Normal	Negative	Negative	Negative	Normal

This improvement continued after she left the hospital, although slight intention tremor in the extremities persisted for a few weeks.

CASE 5.—F. M., 3½ years of age. Eleven days before entry the patient had a brief generalized convulsion, not associated with observed fever. He had another convulsion on the day before admission, and on the day of entry he had three more. There were no other complaints.

On admission he did not appear to be ill and did not show any significant neurologic signs. The deep reflexes were all present and equal and were thought to be hyperactive. On the day after entry he had a brief generalized convulsion. Four days after admission he developed a marked ataxia. He showed no other abnormal neurologic signs and after three weeks in the hospital was discharged to the care of his private physician.

Seven months later he was readmitted for further observation. During the first three months following his discharge he did well, with slight diminution in the ataxia. He then had a resumption of the convulsive episodes. There had been but little change in his condition. The ataxia was still present. A pneumoencephalogram was thought to show a moderate degree of cortical degeneration. His I.Q. was 82. He then began to show moderate myoclonic movements and tremors in addition to his ataxia. During his hospital stay of two weeks all of these symptoms showed some improvement. After discharge there was still further steady improvement. Fourteen months later he appeared to be perfectly normal. His I.Q. at this time was of high average intelligence. There were no longer any convulsions, tremor, or myoclonic movements.

CASE 6.—J. S., 18 months of age. Six weeks before entry this patient had had a high fever of one day's duration. Two weeks before entry he began to have difficulty in walking and it was noted that he had a coarse tremor whenever he attempted to perform a voluntary movement. He fell often but in no particular direction. He soon became totally unable to walk and could not even stand. There had been a few involuntary tremors. He had become somewhat more irritable than usual.

He did not appear to be ill. There was marked ataxia of the head, arms, and body whenever he attempted to perform any voluntary movement. When he was induced to stand, there was a tendency to fall either backward or forward. There was an almost constant movement of the eyes in all directions. The fundi were

TABLE III

SUMMARY OF COURSE IN SEVEN CASES OF ACUTE ATAXIA IN CHILDREN

CASE	AGE	ILLNESS IMMEDIATELY PRECEDING ONSET	ENTRANCE COMPLAINT	CONVULSIONS	RESULT
1	4½ yr.	Acute febrile illness with temperature of 104° F. and sore throat 9 days before admission	Drowsiness, malaise, vomiting, unsteadiness in walking for 3 days	0	Normal 9 months later
2	3 yr.	None	Unsteadiness in walking for 4 days	0	Normal 2 months later
3	2 yr.	None	Unsteadiness in walking for 2 days	0	Died of intraabdominal tumor; neurologic signs almost absent
4	3½ yr.	Upper respiratory infection 6 weeks before admission; irritability and temper tantrums 2 weeks before admission	Clumsiness, indistinct speech, body trembling for 2 weeks	0	Improved
5	3½ yr.	None	Convulsions 11 days before admission	Present	Normal
6	18 mo.	High fever for one day, 6 weeks before admission	Unsteadiness in walking; tremor for 2 weeks	0	Improved
7	2 yr.	None	Dizziness, vomiting, and head tremor for 10 days	0	Marked conduct disturbance

normal. The deep reflexes were hyperactive but were all present and equal. He showed an extreme degree of hypotonia of all the musculature.

The patient was transferred to the Neurological Institute where he remained for twelve and one-half weeks. Although little or no improvement in his condition could be claimed, there was certainly no progression of his disease. On discharge the tremors were still present.

He was seen three months later in the clinic. There had been improvement. The tremors were still present, but he was able to walk again and could pronounce a few words.

CASE 7.—G. S., 26 months of age. Ten days before entry, without any evidence of previous disease, the patient suddenly began to vomit. He also complained of being dizzy, and it was noted that when he held up his head it shook from side to side. When he tried to walk he was unsteady and started to fall but in no particular direction. During the two days before entry he had refused to walk at all.

On admission he appeared to be moderately ill. There was a coarse tremor on all movements of the head and arms and, to a less degree, on movements of the legs. The fundi were normal and there were no abnormal ocular movements, but on later examination the eyes showed an irregular nystagmus. The muscle tone of the extremities was normal. The reflexes were present and equal but were thought to be hyperactive.

After ten days of observation he was transferred to the Neurological Institute with the suspicion that he had a posterior fossa tumor. There, both electroencephalogram and pneumoencephalogram were found to be normal. He was observed for nineteen days, during which time there was little or no improvement in his condition. The day after discharge the family brought him back with the complaint that they were unable to control him at home. The findings were unchanged, but he

was readmitted for a period of sixteen days, during which time there appeared to be little change in his condition other than that the tremor was less prominent, although it was still marked when he became excited. His irritability and restlessness continued so that the parents were unable to control him at home, and he was eventually institutionalized.

The cases presented have certain features in common that delimit them from other disturbances of the nervous system. In all the onset was acute, being ushered in by convulsions in one instance. The outstanding symptoms were those of acute ataxia and muscular incoordination, manifested by intention tremor, which accompanied any movement of the affected part, whether volitional, reflex, or automatic. The slightest attempts at innervation, such as fixation of the eyes, movement of the hand, movements of the extremities, or attempts to speak or smile, in the severe cases, were sufficient to produce tremor. On standing, the general tremor in most patients was increased, the legs trembled, the trunk oscillated, and the head was in constant motion. In a position of rest and muscular relaxation the tremor ceased. Static equilibrium was greatly affected in some cases and less so in others.

In view of the marked body ataxia, nystagmus, with dizziness in one patient, and tremors of the extremities, it is quite possible that there was involvement of the midbrain in such structures as the red nucleus and the brachium conjunctivum. However, no tonic neck reflexes could be demonstrated in the patients showing the most severe symptoms.

In general it may be said that the criteria for purely cerebellar localization are not well established; as a result one usually depends on neighborhood signs for added confirmation. In one of our cases the dizziness suggests a disturbance of the vestibular connection as accounting for the nystagmus.

The appearance of mild myoclonic and choreic features in two of the patients is of special interest. The occurrence of myoclonus in conjunction with a special type of cerebellar or cerebellar pathway disease may have a deeper pathologic significance than would appear. Whether the efferent dentate tract of the cerebellum or the striatum is at fault has not been settled, but it seems quite possible that myoclonic and choreic movements could be due to disturbances which cause synergistic groups of muscles to perform useless or awkward motions. Lesions of the cerebellum and cerebellar connecting tracts might, therefore, produce such symptoms as myoclonus, chorea, and athetosis.

There was a slight increase in cells of the spinal fluid in two of the patients, without changes in the chemical findings. No evidence of increased intracranial pressure developed. A striking characteristic of all of the patients was their clinical improvement, amounting in most instances to complete recovery. In only two patients was the illness followed by a behavior disorder suggestive of a postencephalitic disturbance, and in one of them the change in conduct was comparatively

mild and only temporary in nature. In this respect the favorable course of the majority of the patients in our series contrasts strikingly with our experience in various types of encephalitis in children, where conduct disturbances and personality changes are highly prevalent.

In four of our patients blood serum was tested for neutralizing antibodies against the viruses of St. Louis encephalitis and Eastern equine encephalomyelitis, invariably with negative results. In two of the patients blood sera were also tested for the viruses of Western equine encephalomyelitis, louping ill, lymphocytic choriomeningitis, and herpes simplex, all with negative results likewise. Admittedly there was little in the clinical examination and especially in the spinal fluid to suggest the presence of any of these diseases.

Electromyograms in one severe case, showing head, body, and extremity tremor, demonstrated the tremor to be of cerebellar origin.

With such clinical criteria and in the absence of pathologic confirmation, the identification of the factors responsible for the ataxia and its associated symptoms in our series of cases must be considered unsatisfactory. Several conditions have been recognized which, in some degree, simulate the syndromes which have just been described. Acute infections, trauma, intoxications, or vascular lesions sometimes give rise to acute ataxia, and ataxia may occur as the only manifestation of an encephalomyelitis.[10, 11] This is particularly true of the encephalomyelitis following measles, varicella, and others of the acute exanthemas. Cerebellar ataxia may occur also in association with evidence of widespread lesions of the nervous system following the exanthematous diseases. In some instances, as in our group, the same symptoms developed without any recognizable preceding illness. Such cases have at times been attributed to influenza or to epidemic encephalitis, but the evidence for this association is very unconvincing.

The most important aspect of differential diagnosis in this age group is to distinguish the relatively benign cases of the type described in this paper from those caused by posterior fossa neoplasm. The latter usually begin more insidiously and show a steady or intermittent progression. In almost all instances they manifest evidences of increased intracranial pressure. Cerebellar tuberculoma might simulate this condition. Disseminated sclerosis is a rare cause of ataxia of a cerebellar type in childhood. The symptoms of Schilder's disease may at first be purely cerebellar, although they are usually of a cerebral type. The rare Pelizaeus-Merzbacher disease begins with a cerebellar ataxia and a peculiar "wheel" nystagmus. The hereditary ataxias, including that of Friedreich, as a rule begin insidiously and progress almost imperceptibly.

Some authors consider acute cerebellar ataxia a cerebellar form of poliomyeloencephalitis. It is true that in epidemics of either encephalitis or poliomyelitis overlapping forms are seen, patients with poliomyelitis

exhibiting invasion of the cerebrum, cerebellum, and cranial nerve nuclei, and a few with epidemic encephalitis showing lower motor neurone disease.

SUMMARY

Seven cases of acute ataxia of unknown origin in children are reported. In this group three patients gave a history of acute febrile illness shortly before the appearance of neurologic signs. Four of the patients had been in good health up to the time of appearance of the ataxia. The predominant characteristics of the illness were ataxia and tremor. Convulsions were present at the onset in one patient. Recovery from the neurologic symptoms was complete in five of the patients, and there was very little residual impairment in coordination in the other two. A marked conduct disturbance developed in one patient. We believe that the chief importance of the recognition of this syndrome in this age group lies in its differentiation from cerebellar neoplasm.

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RATBITE FEVER CAUSED BY STREPTOBACILLUS MONILIFORMIS

Case Report

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ALTHOUGH recent investigation of fever following ratbite has tended to emphasize the importance of *Streptobacillus moniliformis* as a frequent etiologic agent, there still remain relatively few reports of cases where this organism has been definitely isolated. Brown and Nunemaker,[1] in a review of cases of ratbite fever reported in the American literature through 1940, listed twelve cases where the *S. moniliformis* was successfully cultured and added three more such cases of their own. In the patient whose case history is to be reported, *S. moniliformis* was cultured from the blood stream on three occasions.

Both the symptomatology and bacteriology of *S. moniliformis* infection are becoming more clearly defined. Perusal of the literature confirms the impression obtained from the study of our own case; namely, that the early symptoms may suggest streptobacillus infection and special media may reveal the organism when routine methods of culture fail.

CASE REPORT

M. C. (146046), a 6-month-old male white infant, was admitted to the pediatric service of the Bronx Hospital, May 19, 1943, with the chief complaint of swelling of the right ring finger following a ratbite five and one-half days previously and fever of six hours' duration.

The parents stated that five and one-half days prior to admission, they had been awakened in the middle of the night by the cries of the patient and had found him bleeding freely from several small lacerations resembling tooth marks over the proximal phalanx of the right ring finger. Near by on the floor the baby's rubber nipple was found apparently chewed up by a rat. Rats had recently been caught in other apartments of the same building. A physician was called; he applied iodine to the wound.

Two days later the injured finger was noted to be moderately swollen. Boric acid soaks were applied, and a total of 16 gr. of sulfadiazine was given. The swelling of the finger diminished somewhat, and there were no other notable symptoms until six hours before admission to the hospital. At this time the patient began to appear irritable and developed fever. The temperature just prior to admission was 104° F. No generalized rash had been observed up to this time, but a few small petechial spots had been noted on the left leg for about twenty-four hours.

About nine days before admission the patient had received his first injection against diphtheria. No local or general reaction had been noted. Birth and development were normal. There was no known exposure to contagion, and there were no previous illnesses.

From the Pediatric Service and the Laboratories of the Bronx Hospital.

On physical examination the infant appeared moderately irritable but in no acute distress. The temperature was 102.8° F. The skin showed a diffuse light macular eruption which was most marked on the extremities. During the next four hours the rash became much more pronounced, rather rubellaform. There were a few petechial spots noted on the left leg. Over the proximal phalanx of the right ring finger there was an area of moderate swelling which was somewhat reddened. In the center of this area were several healed tooth marks suggestive of a ratbite. There were no lymphangitic streaks and no abnormal adenopathy. One small node was palpated in the right axilla, apparently not tender. The joints were normal in appearance and were not tender to palpation or manipulation. The pharynx was slightly reddened. There was slight hyperactivity of the deep reflexes. The heart and lungs and other physical findings were normal.

Laboratory Findings.—Blood count: Hemoglobin, 64 per cent; red blood count, 3,660,000; white blood count, 12,200, with 70 per cent polymorphonuclears and 10 per cent band forms. Subsequent white blood counts were as follows: 16,000, 10,500, 14,800, 13,700, 9,900. Urinalysis was negative except for a very faint trace of acetone and rare white blood cells on one occasion. The blood Wassermann and Kahn reactions were both negative on the fourth and eighth hospital days. Blood serum agglutination reactions were negative for typhoid, paratyphoid, brucellosis, and *Proteus X19*.

Treatment.—Sulfadiazine, 16 gr., had been administered previous to admission. Twelve hours after admission sulfadiazine was started in the dosage of approximately 2 gr. daily per pound of body weight; it was discontinued twenty-four hours later. A three-day course of sulfathiazole was given from the sixth to the ninth hospital days. No other treatment was employed except wet dressings locally and a blood transfusion, 140 c.c., on the seventeenth day.

Course.—As illustrated in Fig. 1, the temperature during the first five days fluctuated between normal and 104.6° F. Following this period, except for a rise to 101.6° F. on the ninth day, the temperature remained essentially normal, with maximum elevation to 100.1° F. (Note the relation to the administration of sulfonamides to be discussed later.)

The patient's general condition remained good throughout the course of his illness. On the third day the generalized rash described previously began to fade and had completely disappeared at the end of a week. Other lesions appeared of a different character. These consisted of nodules on the finger tips and on the side of the nail bed. They were violaceous at the base, papular with a white, apparently pustular, apex. Smears and cultures (aerobic and anaerobic) of the contents of one of these pustules were negative.

At no time was there any evidence of arthritis or other complications. The patient was discharged cured on the twentieth hospital day. Continued observation (up to the thirty-fifth day at the time of writing) has revealed no recurrence of symptoms or signs.

Bacteriology.—A blood culture taken on the day of admission, cultivated aerobically on nutrient agar and in nutrient broth, showed a growth of hemolytic *Staphylococcus albus* after forty-eight hours, twenty-two colonies per cubic centimeter.

On the same day 2 c.c. of the patient's whole blood was inoculated intraperitoneally into a white mouse and an additional 2 c.c. into a guinea pig. The mouse died after twenty-four hours. Smears of the heart's blood showed gram-negative filamentous rods resembling the descriptions of *S. moniliformis*. India ink smears and dark-field examinations revealed no spirilla. The guinea pig remained apparently well and was killed after about two weeks. Smears and dark-field examinations of the heart's blood were negative.

Blood cultures repeated on the fourth and seventh hospital days were grown aerobically and anaerobically under carbon dioxide. Aerobically there was a growth

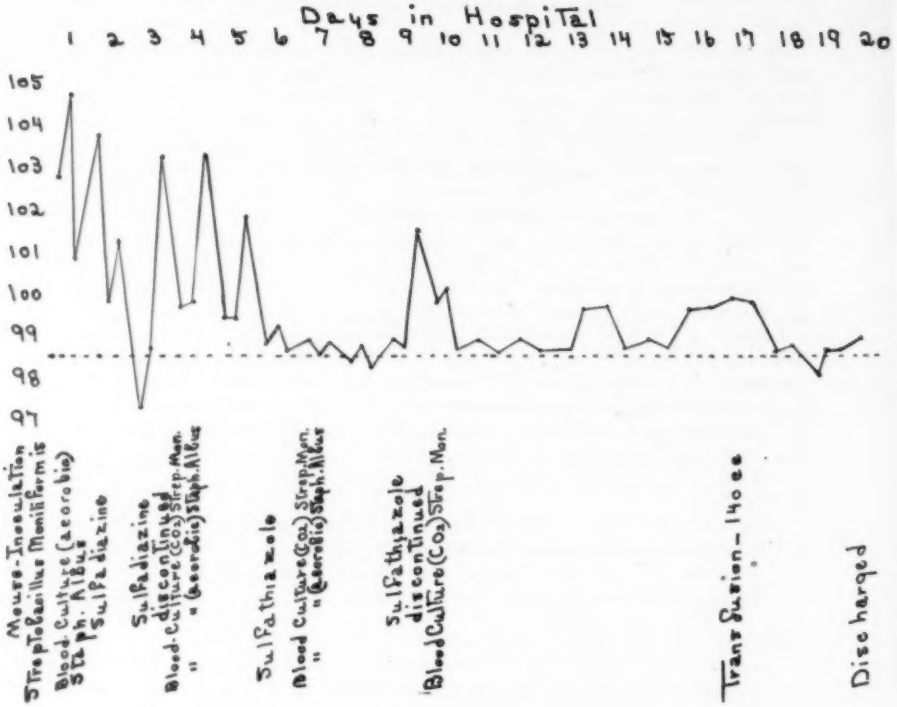
Days in Hospital
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20
105
104
103
102
101
100
99
98
97
Mouse-Inoculation
Streptobacillus Moniliformis
Blood Culture (Aerobic)
Sterile
Sulphadiazine
Sulphadiazine
Blood Culture (Co2) Strep. Mon.
" (Aerobic) Staph. Albus
Sulfathiazole
Blood Culture (Co2) Strep. Mon.
" (Aerobic) Staph. Albus
Sulfathiazole
discontinued
Blood Culture (Co2) Strep. Mon.
Transfusion - 140 cc
Discharged

Fig. 1.

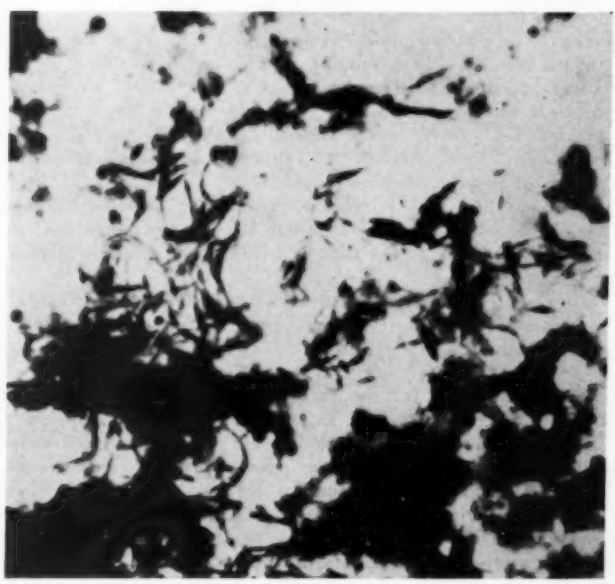

Fig. 2.

on both occasions of hemolytic *Staph. albus* after forty-eight hours. The anaerobic cultures under carbon dioxide showed growths at the end of seventy-two hours of gram-negative filamentous rods, with occasional branching and a tendency to pleomorphism (Fig. 2). This organism corresponded in every respect with the descriptions of *S. moniliformis*. Growths of the same organism were obtained by subculture in various enriched media under carbon dioxide: (a) tryptose broth plus human serum; (b) tryptose broth plus ascitic fluid; (c) tryptose broth plus rabbit's blood; (d) brain-liver-heart semisolid media; and (e) patient's own blood plate.

Two mice were inoculated with portions of an anaerobic broth culture, one intraperitoneally, and one subcutaneously in the thigh. Both mice died in thirty-six hours. In the latter mouse, there was a hemorrhagic area in the muscle of the groin. Smears from this area showed the same streptobacillus described previously. Smears of the heart blood of both mice showed the same organism.

TABLE I

BACTERIOLOGY

I. *May 19—First hospital day*

A. Blood culture—aerobic—nutrient agar and nutrient broth; *Staph. albus*, 22 colonies per cubic centimeter of blood after forty-eight hours' inoculation

B. Whole blood, 2 c.c., from patient injected intraperitoneally in mouse; mouse died after twenty-four hours; smears of heart's blood showed gram-negative filamentous rods

C. Guinea pig inoculated intraperitoneally, killed after two weeks; smears and dark-field examination negative

II. *May 22 and May 25—Fourth and seventh hospital days*

A. Blood culture—aerobic—*Staph. albus* after forty-eight hours

B. Blood culture—anaerobic—usual media—broth showed growth at seventy-two hours of gram-negative filamentous rods

1. *Subculture of II, B*

Tryptose broth + human serum
Tryptose broth + ascitic fluid
Tryptose broth + rabbit's blood
Brain—liver—heart semisolid media
Patient's own blood plate
} Growth, after seventy-two hours of anaerobic incubation, of gram-negative filamentous rods

Subculture of above organism in differential media showed no fermentation of sugars after seventy-two hours of incubation; subcultures on nutrient agar and nutrient broth under anaerobic conditions showed no growth; grown aerobically on these media, a growth of *Staph. albus* was obtained

Subculture on Endoplate aerobically showed no growth

2. Anaerobic broth culture (II, B) inoculated into three mice, two intraperitoneally, one subcutaneously into thigh; mouse injected into thigh died in thirty-six hours; one of the mice injected intraperitoneally died in thirty-six hours; the other was sacrificed on twentieth day

(a) Mouse injected intraperitoneally—smears of heart's blood showed gram-negative filamentous rods

(b) Mouse injected subcutaneously in thigh:

(1) Heart's blood smear—gram-negative filamentous rods
(2) Smear of hemorrhagic area in muscle of groin—gram-negative filamentous rods

(c) Mouse injected intraperitoneally killed twenty days later Smears of heart blood showed *S. moniliformis*

III. *May 28—Tenth hospital day*

Blood culture (anaerobic)—*S. moniliformis*
Subculture identified by National Institute of Health as *S. moniliformis*

A third mouse inoculated intraperitoneally with an anaerobic broth culture showed no symptoms. However, when this mouse was killed twenty days later, smears of the heart's blood showed *S. moniliformis.*

Anaerobic blood culture under carbon dioxide on the tenth hospital day again revealed *S. moniliformis.* A subculture of this was sent to the National Institute of Health, where Dr. Carl H. Larson identified the organism as *S. moniliformis.* (Table I.)

COMMENT

Although there appears to be no doubt as to the identity of the *S. moniliformis* isolated in this case, the clinical picture was somewhat confused by the finding of *Staph. albus* in aerobic blood cultures. Several considerations militate against the theory that this was merely a contaminant. In the first blood culture there were twenty-two colonies per cubic centimeter, a more profuse growth than one would ordinarily ascribe to a contaminant; the organism was found in blood cultures taken also on the fourth and seventh hospital days, but it was absent on the tenth day. (In the interim sulfathiazole had been given.) The organism was an hemolytic form of *Staph. albus;* no further tests for virulence were made at that time. Tests for anticoagulase in the patient's serum thirty-five days after the ratbite were performed by Dr. Gregory Schwartzman; these tests were negative. Furthermore the patient was only 6 months of age, a favored period for staphylococcus septicemia. Thus there seems to be fairly strong evidence of a double infection, with *S. moniliformis* and *Staph. albus.*

The temperature curve, the administration of sulfonamides, and the probable double infection present an interesting but difficult problem to analyze (Fig. 1). From the sixth to the ninth hospital days, a period of normal temperature, sulfathiazole was given; during this time, on the seventh day, blood culture still showed the presence of *Staph. albus.* On the tenth day, one day after the discontinuance of sulfathiazole, blood culture was still positive for *S. moniliformis.* There are no reports of definitively beneficial results from the use of sulfonamides in *S. moniliformis* infections; on the other hand, the response of staphylococcus infections to this treatment is well known. With these thoughts in mind one is inclined to ascribe the fever to the combined infection and the persistence of *S. moniliformis* in the blood to its resistance to sulfathiazole therapy.

The paucity of reports of authentic cases warrants emphasis on any symptoms that might be of diagnostic value. The nodular cone-shaped lesions, capped with yellow heads, previously described, seem similar to the lesions referred to by Allbritten and co-workers[2] and possibly also the lesions described by Farrel and his co-workers.[3] These lesions were much firmer and more nodular than the pustular or bull's eye lesions characteristic of staphylococcus infections. Possibly one should not emphasize their finer characteristics but rather their petechial nature; the exact characteristics of petechiae may vary with the kind of tissue

affected, the age of the patient, and their location at the extremities. Nevertheless it would be interesting to know whether such lesions occur more or less regularly in streptobacillus infections.

GENERAL DISCUSSION

For a complete review of the subject of ratbite fever, the reader is referred to the articles of Farrell, Lordi, and Vogel,[3] Allbritten, Sheely, and Jeffers,[2] Kato,[4] and particularly to the comprehensive review by Brown and Nunemaker.[1] It is now fairly well established that fever following ratbite may be caused by either of two etiologic agents, the *Spirillum minus* and the *S. moniliformis* (also commonly known as *Haverhillia multiformis*). These two organisms biologically so different produce similar clinical pictures. Sodoku, the disease caused by *Spirillum minus,* is produced only by the bite of a rat or other animal carrying the organism. On the other hand, *S. moniliformis* septicemia is caused by a ratbite and also follows the ingestion of contaminated food. The latter disease has been called Haverhill fever.

The clinical picture of ratbite fever due to the *Spirillum minus* is as follows: There is a history of a bite by a rat, followed by an incubation period of one to four weeks. There then follows a flare-up of the original wound with a marked local inflammatory reaction. Lymphangitis is present and the lymph nodes draining the area are enlarged and tender. There is frequently a bluish red maculopapular eruption which may be generalized but is often restricted to the area about the wound. Recurrent paroxysms of fever occur of a remittent or intermittent type. The blood Wassermann reaction and particularly the Kahn reaction may be positive. The specific treatment is one of the arsenicals; the response is typically good, with abrupt cessation of fever and symptoms. Recurrences may occur, necessitating repeated doses of arsenicals.

The *Spirillum minus* was isolated from such cases by Fataki in 1917. It is a highly motile spirillum, often with one or more flagellae. It may be visualized under dark-field examination and with Giemsa or silver stains. The organism has been discovered by dark-field examination of the fluid from the local lesions or from the affected lymph nodes of patients and has been rarely reported to have been visualized in smears of the peripheral blood. Diagnosis is usually made by the inoculation of whole blood of the patient into white mice and guinea pigs, with repeated study of peritoneal exudate, blood, and inguinal glands of these animals. There are no accepted reports in the literature of the successful artificial cultivation of this organism.

The historical development of knowledge pertaining to the *S. moniliformis* was summarized in 1939 by Farrell, Lordi, and Vogel[3] and by Brown and Nunemaker.[1] Knowledge of this organism dates from 1914, when Schottmüller, investigating a case of fever following a ratbite, isolated an organism by blood cultures which he termed *Streptothrix*

muris ratti. Reports of similar organisms in ratbite fevers followed in 1916, 1917, 1918, 1925. In 1925, Levaditi, Nicolau, and Poincloux recovered an organism similar to the one just mentioned from a patient with remittent fever with a maculopapular rash and arthritis, not associated with ratbite. They named this organism *S. moniliformis*. Place, Sutton, and Willner,[5] in 1926, investigated at Haverhill, Mass., an epidemic of a similar type of fever accompanied by rash and arthritis. The bacteriology of this epidemic was reported in detail later in the same year by Parker and Hudson.[6] These cases were not associated with ratbites. The source of the infection was traced to a dairy distributing unpasteurized milk. The authors believed that this was a new disease and named it erythema arthriticum epidermicum. From ten patients an organism similar to those mentioned previously was isolated by blood culture. In consideration of the locations of the epidemic and because of the pleomorphic nature of the organism, it was named *Haverhillia multiformis*. It is now felt that all these organisms reported independently are probably the same. Brown and Nunemaker prepared a list of the many names applied to this organism; even now it is not quite certain whether it has acquired a permanent name and achieved legitimacy. *S. moniliformis* seems to be the generally accepted title.*

The discovery of the identical organism both from cases due to ratbite and from cases apparently due to the ingestion of contaminated food has caused considerable confusion. Aside from the local lesion, the symptoms are the same. Both conditions should be thought of as *S. moniliformis* septicemia with different portals of entry of the infecting agent.

The disease is characterized by an abrupt onset with chills, fever, vomiting, and headache usually following an incubation period of about three to six days; in some cases the incubation period is longer, indeed as long as in Sodoku, the spirillary type of ratbite fever. There is characteristically an early maculopapular rash frequently resembling rubella present mostly on the extremities. Arthritis is typically present, often severe and involving many joints. A characteristic fever curve has been described rising abruptly at first with remission in from two to five days, then followed by a recurrence with arthritic symptoms. The serologic tests for syphilis as a rule are negative. The white cell count may be normal but usually is elevated. No successful specific treatment has been established, although some good results have been reported with thymol. Usually there is no response to arsenicals, and sulfonamides are reported to be of no value. The prognosis, however, is good.

*The literature available to us under present wartime conditions since the review of Brown and Nunemaker in 1940 reveals one interesting case of streptobacillus infection following the bite of a rat.[10] This occurred in a 13-year-old girl with chronic rheumatic cardiac disease. Blood cultures on "ordinary media" were negative. The patient died. At post-mortem, cultures of the valvular vegetations in "ordinary media" were also negative. Injection of the material into the foot pads of mice and subsequent culture on enriched media revealed the *S. moniliformis*.

TABLE II

DIFFERENTIAL CHARACTERISTICS OF SPIRILLUM MINUS AND STREPTOBACILLUS MONILIFORMIS SEPTICEMIA

SPIRILLUM MINUS	STREPTOBACILLUS MONILIFORMIS
History of ratbite (or other animal bite)	May or may not have history of ratbite
Long incubation period, from one to four weeks	*Short* incubation period significant; it may be long
Recurrence of local lesion with each onset of fever	Local lesion at onset of disease
Marked local lymphangitis and lymphadenitis	Mild adenopathy or none
Bluish red maculopapular rash occurs in some cases	Erythematous maculopapular rash occurs in most cases
Myalgia; no arthritis	Arthritis usually present
Serologic tests for syphilis frequently positive, particularly flocculation test (Kahn)	Serologic tests for syphilis rarely positive
Good response to arsenicals	No established specific treatment
Animal inoculation: Spirilla in blood stream	Animal inoculation: frequently arthritis and death; *S. moniliformis* in heart's blood and local lesions
Agglutination tests not of definite value	Agglutination tests usually positive*
Blood culture negative	Blood culture positive

*In the chart prepared by Allbritten, Sheely, and Jeffers[3] and included by Kato in his chapter on Ratbite Fever in Brennemann's *Practice of Pediatrics*,[4] it is stated that "precipitin and complement fixation (tests) offer confirmatory evidence." We could find no record of positive precipitin and complement fixation tests *in human beings*. In a personal communication, Dr. Carl Larson of the National Institute of Health wrote: "Precipitin and complement fixation tests have not been used in this laboratory and, according to other workers, it is probable that neither of these tests yield results comparable to those given by agglutination tests."

Although there is much overlapping of symptoms in the spirillary (Sodoku) and bacillary forms, suggestive diagnostic criteria do exist. These are outlined schematically in Table II. Some of these criteria deserve special comment. Thus, a short incubation period, as in the case being reported, is extremely suggestive of streptobacillus infection. However, an incubation period of two weeks or more is of no significance, occurring regularly in Sodoku and not infrequently in *S. moniliformis* disease. Arthritis is a most important symptom; when present it is quite positive evidence of streptobacillary infection rather than Sodoku. In the latter disease it does not occur at all or is an extreme rarity. Brown and Nunemaker[1] and Larson[7] have considerable confidence in the validity of the agglutination tests for streptobacillus disease; they consider a test positive in high dilution evidence of a previous infection in cases where there is no record of a positive blood culture.

The bacteriologic aspects of the disease will not be discussed in detail by us; this can be found in the reviews referred to and in Topley and Wilson's textbook.[8] As noted in Fig. 2 the organism isolated in our case corresponds to the description of Parker and Hudson: "Slender, gram-negative and nonacid resisting rods staining with some difficulty; often forming threads and showing tendency toward branching; marked irregularity of form with swellings and enlargements; fermentation of some carbohydrates; in general, requiring blood or

ascitic fluid for growth." From a practical standpoint, the varying and, at times, contradictory results of blood culture "on ordinary media" should be emphasized. Hazard and Goodkind[9] obtained the organism from "5 to 10 c.c. of blood in 100 c.c. of plain broth (beef infusion containing 0.1 per cent dextrose)." On the other hand, Brown and Nunemaker write "The methods for the cultivation and study of the streptobacillus represent a definite departure from our usual concepts of complete clinical bacteriologic methods." Again these authors, in referring to their own three cases, state: "In each instance the clinical bacteriologic laboratory reported negative blood cultures, whereas parallel cultures in which the special techniques were used yielded abundant growth of the microorganism." The present case report confirms that viewpoint; the blood culture became positive only when the anaerobic technique was used. Complete protocol of bacteriologic studies is to be found in Table I.

A more or less standardized approach to the study of a case of ratbite fever seems now available, and the use of special techniques no doubt will reveal a greater number of cases due to the spirillum and to the *S. moniliformis.**

SUMMARY

1. A case of *S. moniliformis* septicemia following ratbite is presented with clinical and laboratory findings. The additional isolation of *Staph. albus* and the effect of sulfonamides in this case are discussed.

2. The subject of ratbite fever is briefly reviewed.

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*Several cases of mixed spirillary and streptobacillus moniliformic infections have been reported; Brown and Nunemaker question the adequacy of the evidence presented. Such mixed infections theoretically are possible and studies should be made for both organisms.

TREATMENT OF PNEUMONIA WITH A SINGLE DOSE OF SULFADIAZINE

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SHORTER administration of sulfonamides may be expected to decrease the risk of toxic manifestations, the sensitization to the drug, and the cost of the treatment. Therefore we investigated whether a single dose of sulfadiazine acts as promptly in children with pneumococcus pneumonia as the conventional prolonged course of chemotherapy.

Alternate children admitted with pneumonia were treated with single doses of sulfadiazine while the others were treated with a regular course of sulfadiazine. Age distribution and duration of pneumonia before admission were approximately the same in the children in both groups.

The group in which a single dose was given consisted of twenty-five children, the ages ranging between 8 months and 10 years. Blood counts and urine examinations were done before the single doses were given and three days later. Drug levels in the blood were determined on the morning following the day the drug was administered. Consequently the intervals between the administration of the drug and the determination in the blood varied between six and twenty-four hours, and the figures obtained did not indicate the maximal drug concentration in the blood. With one exception, sulfadiazine was given by mouth in a single dose of not less than 0.15 Gm. per kilogram of body weight. Smaller children weighing up to 20 kg. received relatively higher doses, from 0.2 to 0.3 Gm. per kilogram of body weight. The maximal dose was 5.0 Gm. of sulfadiazine. Fluid was forced during the days after the administration of the drug, but no sodium bicarbonate was given.

Those in the control group, consisting of twenty-three children with pneumonia, were treated during the same winter (1942-1943) with 0.2 Gm. of sulfadiazine per kilogram of body weight for the first day, followed by 0.1 Gm. per kilogram of body weight daily in divided doses and continued until the temperature was normal for two days.

RESULTS

The effect of single doses of sulfadiazine is shown in Table 1. Of twenty-five children with pneumonia, three cases (10, 21, and 24) were complicated with otitis media and the children did not respond to the single dose treatment; fever continued for five and one-half, six, and eight days, respectively, and then subsided. The single dose treatment seems to be inadequate for pneumonia complicated with otitis.

From the Department of Pediatrics of the Lincoln Hospital.
Sulfadiazine was supplied by the Lederle Laboratories, Pearl River, N. Y.

TABLE I

AGE	WEIGHT (POUNDS)	DURATION OF PNEUMONIA (DAYS)	DOSE OF DRUG (GM.)	BLOOD LEVEL (MG.)	W.B.C. ON ADMISSION	W.B.C. THREE DAYS LATER	ROENTGEN FINDINGS	DURATION OF FEVER AFTER SULFADIAZINE	TEMPERATURE ON ADMISSION (° F.)	RECURRENCE OF TEMPERATURE	URINE ON ADMISSION	URINE 3 DAYS LATER	CASE
8 mo.	20	1	2.7	Tr.	12,500 (86% P.)	9,700 (80% P.)	---	12 hr.	103.6	No	Occas. W.B.C.	Occas. W.B.C.	1
10 mo.	18	1	2.5	7.7	21,500 (86% P.)	15,000 (80% P.)	R.L.L.	4 hr.	100.8	No	Several W.B.C./hpf	Occas. W.B.C.	2
13 mo.	16	1	2.5	4.7	11,500 (78% P.)	24,200 (76% P.)	R.U.L.	2 days	104.0	No	1 W.B.C. tr. alb.	Neg.	3
1½ yr.	25	2	3.4	4.6	16,000 (90% P.)	9,000 (75% P.)	R.U.L.	14 hr.	105.6	No	Neg.	Occas. R.B.C.; gran. cast.	4
20 mo.	23	4	3.0	1.8	22,000 (87% P.)	12,000 (75% P.)	R.M.L.	3 days	105.2	No	Tr. alb.	Neg.	5
2 yr.	23	3	3.0	7.5	20,000 (85% P.)	9,000 (70% P.)	R.M.L.	3 days	103.8	No	Neg.	Neg.	6
2 yr.	22	6	3.0	8.6	19,400 (76% P.)	12,500	R.L.L.	14 hr.	104.5	No	5 W.B.C.	6 W.B.C.	7
2 yr.	24	10	3.0	1.4	17,800 (90% P.)	14,000 (90% P.)	L.L.L.	16 hr.	104.0	No	Neg.	Neg.	8
2½ yr.	24	6	3.0	1.9	30,000 (88% P.)	15,200 (60% P.)	R.L.L.	4 hr.	104.0	No	Tr. alb.	Neg.	9
2½ yr.	27	4	3.7	5.1	18,600 (80% P.)	12,000 (72% P.)	L.L.L.	8 days*	104.2	No	Neg.	Neg.	10

2½ yr.	28	7	3.5	8.4	14,200 (85% P.)	7,500 (65% P.)	R.U.L.	†	103.0	No	Neg.	Neg.	11
3 yr.	33	3	3.0	4.1	21,400 (72% P.)	6,800 (60% P.)	L.L.L. R.L.L.	†	104.0	No	Neg.	Neg.	12
3 yr.	32	5	3.0	6.7	27,000 (90% P.)	10,000 (73% P.)	L.L.L.	>3 days‡	104.0	Yes	Tr. alb.	Neg.	13
3 yr.	32	3	3.0	3.7	27,000 (95% P.)	9,000 (84% P.)	L.L.L.	12 hr.	104.4	No	Tr. gluc.	Neg.	14
3 yr.	33	2	3.0 i.v.	19.2	14,000 (85% P.)	11,000 (86% P.)	R.U.L.	2 days	106.0	Yes	Neg.	Neg.	15
4 yr.	44	4	3.6	2.1	12,800 (82% P.)	10,000 (80% P.)	L.L.L.	30 hr.	104.8	Yes	Neg.	Neg.	16
4 yr.	41	4	3.8	2.4	32,000 (90% P.)	6,650 (31% P.)	R.U.L.	20 hr.	103.5	No	Neg.	Neg.	17
4 yr.	35	9	4.0	1.5	38,400 (82% P.)	40,000 (62% P.)	R.L.L.	3 days	101.4	No	Tr. alb.; 10 W.B.C./hpf	Tr. alb.; 3 W.B.C./hpf	18
4 yr.	41	3	4.0	2.4	9,500 (90% P.)	9,000 (80% P.)	R.L.L.	4 days	102.0	No	Neg.	Neg.	19
6 yr.	45	3	3.0	1.3	28,500 (95% P.)	10,800 (67% P.)	R.L.L.	36 hr.	102.5	No	Occas. W.B.C. tr. alb.	Neg.	20
6 yr.	55	1	5.0	2.3	10,200 (85% P.)	10,000 (80% P.)	R.L.L.	6 days*	104.0	Yes	Neg.	Neg.	21
8 yr.	50	2	3.5	0	26,450 (98% P.)	10,000 (70% P.)	R.M.L.	56 hr.	104.0	No	Tr. alb.	Neg.	22
8 yr.	67	4	4.5	3.0	25,000 (90% P.)	10,000 (75% P.)	R.L.L.	20 hr.	105.2	No	Neg.	Neg.	23
9½ yr.	54	7	3.7	4.0	13,000 (80% P.)	14,000 (90% P.)	L.L.L.	5½ days*	101.2	No	Tr. alb.; 6 WBC/hpf	Neg.	24
10 yr.	76	4	5.0	2.0	9,500 (85% P.)	7,000 (75% P.)	R.L.L.	3½ days	102.0	No	Neg.	Neg.	25

*Case of pneumonia complicated with otitis media.
†Temperature does not become normal and remains around 100° F.
‡Treatment with sulfadiazine resumed after three days.

One child (Case 13) with severe bilateral pneumonia still had fever after three days, and a full course of sulfadiazine treatment had to be resumed.

In two other children (Cases 11 and 12) the temperature dropped only to 100° F. and remained at this level throughout the stay in the hospital. Clinically these two children made a quick recovery and did not appear ill.

The remaining nineteen children showed a clinical recovery not different from that of those in the control group. The temperature dropped to normal after from four to ninety-six hours, or an average of 38.4 hours.

In the control group receiving a full course of sulfadiazine the temperature lasted from six to 100 hours after chemotherapy was instituted. Average duration of temperature in these twenty-three children was 40.1 hours.

None of the children receiving the single dose showed any toxic manifestation, though relatively large doses were given, and the urine was not alkalized by administration of sodium bicarbonate.

DISCUSSION

Similar attempts at a single dose treatment of pneumonia in children have been reported by Karelitz and Weinstein[1] with sulfapyridine and by Friderichsen and Søbye[2] with sulfathiazole.

Karelitz and Weinstein[1] administered a single dose of 0.1 Gm. of sulfapyridine per kilogram of body weight to twenty-eight children. There was no effect in two children with empyema. Of the remaining twenty-six children, fever recurred in 20 per cent so that treatment had to be resumed. In another 20 per cent, low-grade fever recurred which did not require more sulfapyridine. Another group of nine children with uncomplicated pneumonia received two doses of sulfapyridine each, 0.1 Gm. per kilogram of body weight, at an interval of twelve hours. All responded promptly, the temperature dropping to normal in from eight to twenty-eight hours, or an average of 18.1 hours.

Friderichsen and Søbye treated seventy children with pneumonia with single "shock" doses of sulfathiazole and fifty-nine children with multiple doses of this drug for four days. The simpler administration of the single large dose was as effective as the treatment with multiple doses, caused fewer secondary toxic effects, and was half as expensive as the continuous treatment. The "shock" dose of sulfathiazole given children weighing up to 13 kg. was 0.3 Gm. per kilogram of body weight, while children weighing 20 kg. received 0.2 Gm. and those weighing 30 kg. received 0.14 Gm. per kilogram of body weight; the maximal dose was from 4 to 5 Gm.

Our series of children receiving similar doses of sulfadiazine seems to confirm the observations of Friederichsen and Søbye with sulfa-

thiazole. The smaller single doses of sulfapyridine used by Karelitz and Weinstein appear inadequate for the treatment of even uncomplicated pneumonia. If these relatively small doses of sulfapyridine were repeated, amounting to a similar total used by Friderichsen and Søbye as well as by us, Karelitz and Weinstein observed prompt results.

Complications of pneumonia such as otitis media and empyema do not respond to single doses of sulfonamides; therefore, single doses of sulfonamides should not be used in complicated cases.

The data available do not suffice to recommend the single dose treatment of pneumonia as the method of choice. There is no need for it in hospitalized children. A full course of chemotherapy appears safer, particularly in preventing complications. However, in ambulatory cases or in a home where nursing care appears inadequate or unreliable, it might be an advantage to resort to the single dose treatment. A full course of chemotherapy may be resumed if the temperature does not drop to normal within forty-eight hours or if the general condition of the child appears serious.

SUMMARY

Uncomplicated pneumococcus pneumonia in children seems to respond as promptly to a single dose of sulfadiazine as to continued doses of this drug.

The temperature dropped to normal in from 4 to 96 hours, or an average of 38.4 hours, following the single dose, and in from six to 100 hours, or an average of 40.1 hours, when a full course of sulfadiazine was given.

No toxic manifestations appeared in twenty-five children following a single dose of from 0.15 to 0.3 Gm. of sulfadiazine per kilogram of body weight, not exceeding a maximal dose of 5.0 Gm. of sulfadiazine.

The single dose treatment is simpler, less expensive, and may decrease the risk of toxic manifestations and the sensitization to the drug.

Three patients with pneumonia complicated by otitis media did not respond to the single dose treatment.

The single dose treatment is not recommended for hospitalized, serious, or complicated cases of pneumonia but as an expedient for ambulatory cases or occasions where proper care is lacking.

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CHONDRODYSTROPHIA CALCIFICANS CONGENITA

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AN UNUSUAL case of congenital maldevelopment was first observed by one of us (M. P. B.) Jan. 11, 1943, ten days after delivery by breech extraction.

This male infant (W.) breathed spontaneously and revealed nothing worthy of note during its stay in the nursery. Birth weight was 7 pounds, 6 ounces, and on leaving the hospital the weight was 6 pounds, 14 ounces.

At this first examination marked cyanosis was observed while crying. A loud systolic murmur was heard over the entire cardiac area which led to the diagnosis of a defect in the interventricular septum. The knees were flexed to a 30-degree angle—more marked on the left leg which could not be extended. These findings in the lower extremities were not considered incompatible with changes observed following breech delivery.

Examination two weeks later revealed an aggravation of the knee findings which necessitated x-ray studies. The films were studied by one of us (J. A.), whose description of the roentgen findings follows:

Feb. 2, 1943, lateral films taken of the left knee (Fig. 1) showed an abnormal calcification which filled the entire upper patellar recessus and extended downward to the interarticular space. While this deposit was coherent like a wax imprint of the entire capsule space, we saw posteriorly many more round or sometimes irregular calcified bodies. A few stippled shotlike densities were found in between but were not as prevalent as at the foot and wrist. There was a periosteal elevation along the anterior surface of the femur. The right knee joint showed similar changes (Fig. 2) March 12, 1943. There were onion-peel-like periosteal elevations (Fig. 3) along both femora and slight periosteal (Fig. 4) thickening along the tibiae. The proximal epiphyses of both humeri were normally well developed. Some stippled densities were seen underneath the epiphyseal center of the humerus. X-ray of the chest showed a markedly enlarged heart and an enlarged thymus.

March 7, 1943, there were multiple stippled calcareous deposits in the distal row of the tarsal bones. We could not call them stippled epiphyses since the proximal and distal epiphyses of the tibia and femur were well developed and the astragalus and os calcis were of normal

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density and showed no stippling. There were similar changes in both wrists (Fig. 5). The area of the knee joints showed a different type of calcification. We saw here on both sides multiple round and partially sickle-shaped calcifications.

The patellar and epiphyseal cartilage was outlined as a negative density by the surrounding calcium wall. The capsule appeared thickened and filled out with the numerous irregular calcifications. It was noted that the distal femur epiphysis was normal but slightly smaller on the left side. The bone structure of the shafts and the epiphyseal lines appeared normal. There was a slight flaring of the metaphyseal ends of the tibia and femur.

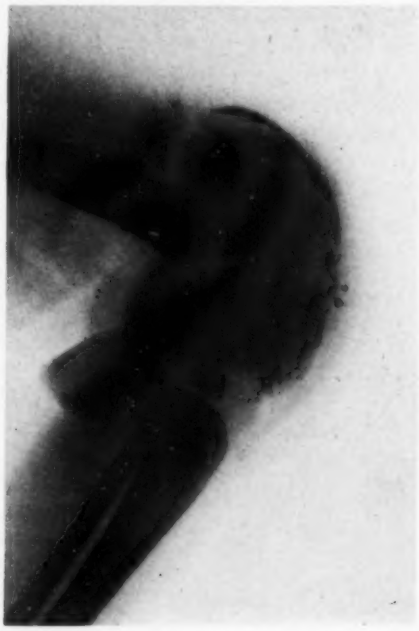

Fig. 1.—*Left Knee:* The upper patellar recessus and capsule are filled with irregular calcifications. The wax imprint of the entire capsular space and multiple round calcified bodies are within the joint space.

The findings were so bizarre and unusual, resembling nothing ever seen by either of us or many of our colleagues, that a definite diagnosis was sought. A search of the literature revealed the relative scarcity of this type of case. Most reports appeared in the radiologic literature.

Raap[1] used the name chondrodystrophia calcificans cogenita to describe this entity. His article contains a comprehensive review of the current literature on this subject with a complete bibliography. The first to describe this condition is thought to have been Conradi[2] in 1914. He found no reports of any similar case in the literature. His roent-

gen findings were as follows: "The changes were most marked in the distal ends of the femurs and in the bones with peculiar shotlike shadows; the distal tibial and fibular epiphyses were affected also while the upper tibial and fibular epiphyses were only slightly involved."

Fig. 2.—*Right Knee:* Similar to Fig. 1. Puttylike material fills the upper recessus and the capsular space. Note numerous stippled densities, usually called "stippled epiphyseal centers." The developed epiphyseal centers, however, are normal in appearance and surrounded by extensive cartilage.

The rarity of this condition is evidenced by the personal report from the Mayo Clinic which states that only one such case was seen there and the x-ray films of a second case. In the Cook County Children's Hospital one similar case was the subject of study. One of the earliest articles

was by Tisdall and Erb[3] who, in 1924, described a 5-week-old infant showing calcareous deposits of many joints. This was most marked in the patella which was cartilaginous and contained loose connective tissue with degenerated cells and these spaces filled with dense calcareous material. This patient, as did ours and the one at Cook County Children's Hospital, had a congenital heart lesion. No abnormalities of the glands of internal secretion were discovered post mortem. Bloxsom and

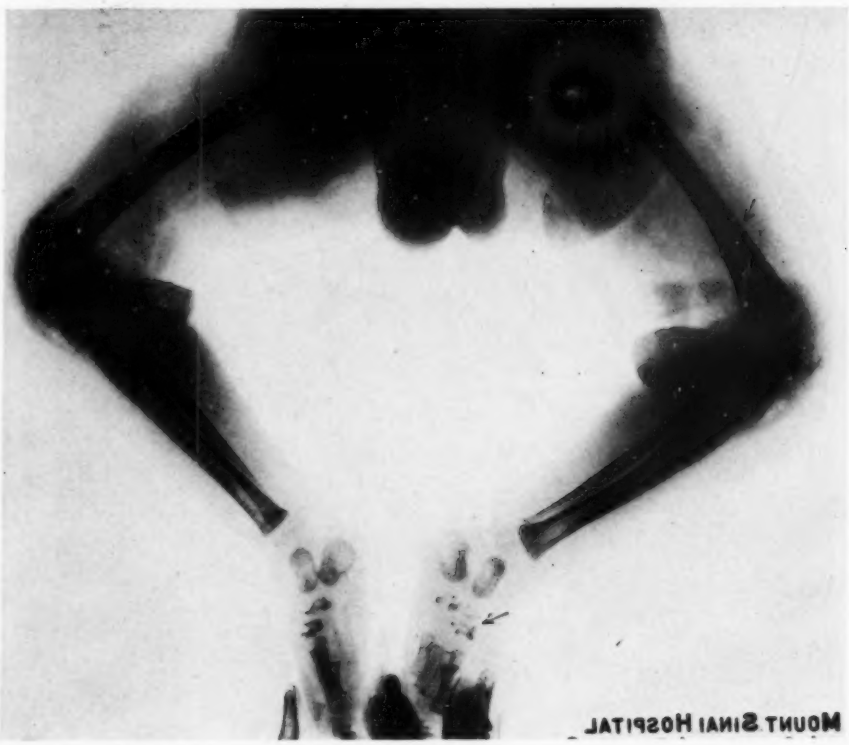

Fig. 3.—Demonstrates bilateral periosteal elevation along the femura and the tibia and stippled calcifications in the region of the tarsal bones. No stippling in the astragalus and os calcis.

Johnston's[4] patient showed calcium deposits in many joints, chest wall, along vertebral column, pelvic bones, and upper and lower ends of the left femur. This patient also showed marked hyperkeratosis and bilateral cataract. The general condition of the patient improved spontaneously. The authors' conclusions were: "This suggests that some disturbance in the placenta or in the calcium metabolism of the mother during gestation overloaded the infant's blood with calcium which was then deposited in the various regions of the body."

In 1931 Hunerman[5] reported a case in a 3-week-old female which coincides closely with our findings. There were stippled shadows in the epiphysis of the extremities as well as along the spinal column and in the pelvis. The skull and facial bones were unaffected. These stippled

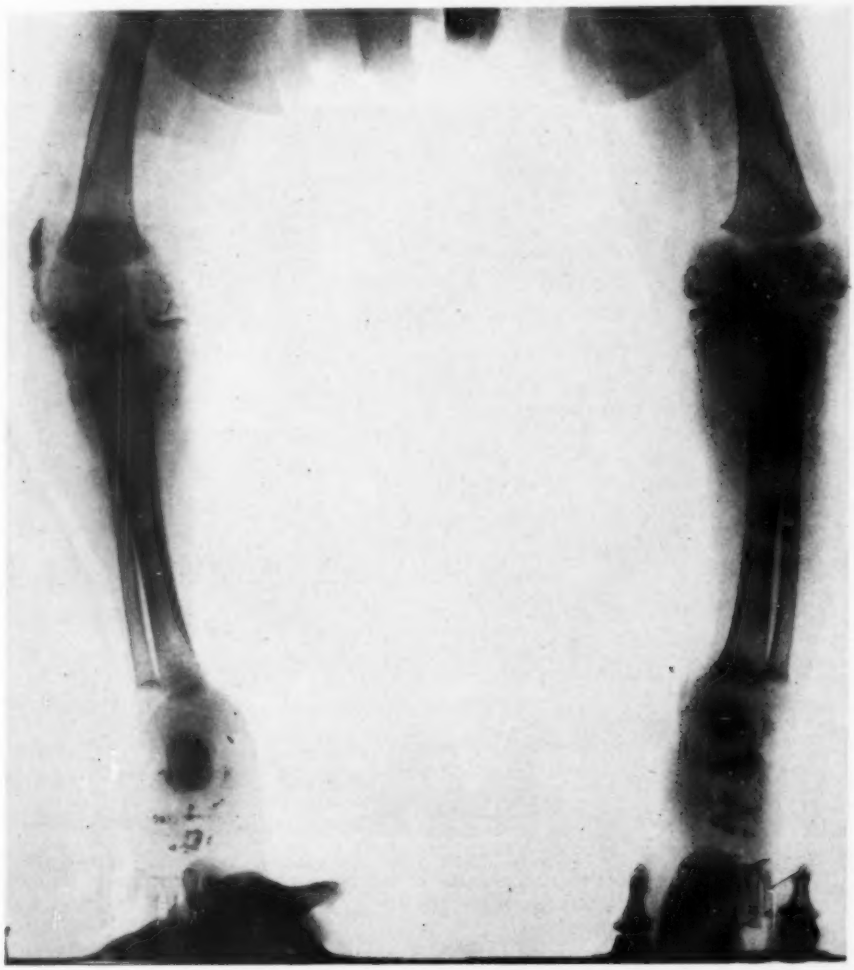

Fig. 4.—Anteroposterior view of both knees shows the relation of the calcareus deposits to the well-developed epiphyseal centers and to the cartilage (translucent area). Spur formation at the inner side of the distal end of femur and periostitis.

shadows were found at the epiphyseal ends of the humeri, radii, ulna, femora, tibiae, ankle joints, and carpal bones. The bony structure of the shafts was normal. These added to our findings of large irregular dense deposits of calcium in both knee joints furnish the reason for the name chondrodystrophia calcificans congenita.

Geyman[6] reported a case in a 2-month-old infant which he considered to be a form of chondrodystrophy. Syphilis he feels is ruled out by the clean-cut appearance of the diaphysis.

Cases of stippled epiphyses in older children that were at first considered to be Perthes' disease were reported by Reilly and Smyth.[7] They are of the opinion that these are due to hypothyroidism because of the clinical reports of marked improvement on thyroid therapy. They also noted the change to a homogeneous appearance of the epiphyses

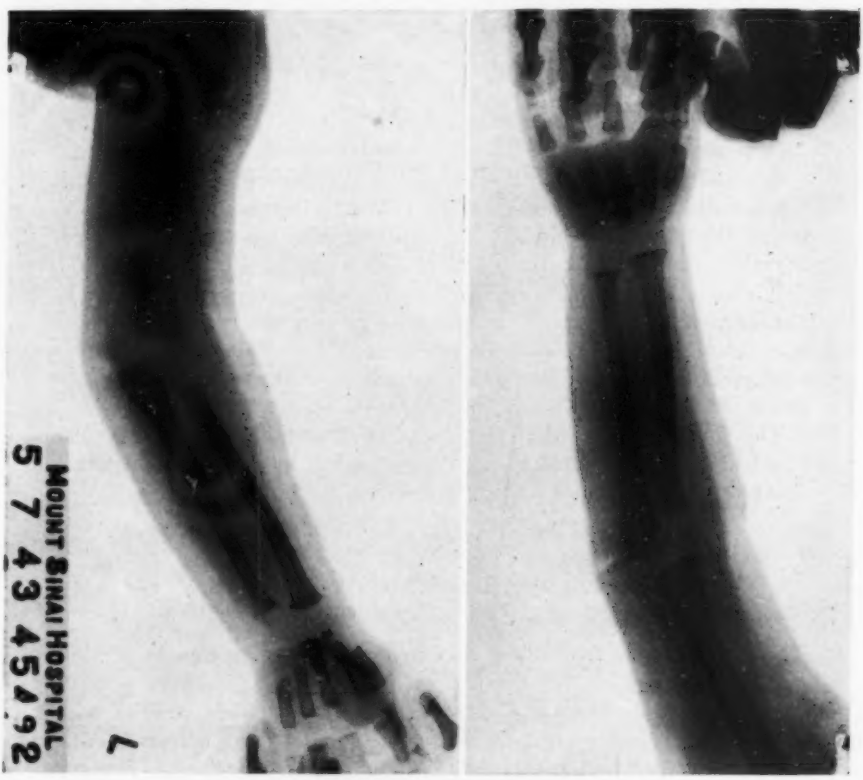

Fig. 5.—No "stippling," no periosteal reaction at the elbow joint, but marked "stippling," pseudoepiphyseal calcification in the wrist bone.

after having earlier been found to be stippled. The appearance of the other epiphyses at the normal time was observed. Siegert,[8] reporting a case of pseudoepiphysis in an athyreotic child, showed in a series of x-ray films the formation of the pseudoepiphysis as a form of chondroplastic osteogenesis from the beginning to complete restitution under thyroid medication.

Under the term osteogenesis chondroplastica Siegert[8] collects all forms of abnormal bone formation of the cartilaginous preformed skeleton. He divides the products of chondroplastic osteogenesis into three groups:

1. Pseudoepiphyseal ossification. Occurrence of epiphyseal-like bone in normally cartilaginous areas early, with or without connection with the diaphysis.

2. Several ossification centers instead of one originating in cartilage on the surface of ossified cartilage and separated from it. The ultimate result is normal bone formation or accessory bones.

3. Pseudoepiphyses of the phalanges.

In 1939 Maitland[9] reported two cases of punctate epiphyseal dysplasia in the same family. The knee and elbow joints were enlarged and flexed. Most marked changes were in the hip, knee, shoulder, and elbow joints. The vertebrae and ribs were also affected.

Case reports of a mother and her two identical twins who showed epiphyseal stippling but no calcium deposits were made by Resnick.[10] The condition was first considered to be Perthes' disease. He decries the use of the name cretinoid epiphyseal dysgenesis. No evidence of hypothyroidism was found in these patients.

A clinical entity, epiphyseal dysgenesis, which some considered to be the diagnosis in our case, is thought to be on a hypothyroid basis. There was no evidence of this condition in our patient. There was no history of thyroid disturbance in the mother and no clinical or laboratory evidence of thyroid deficiency in the infant.

The development of the carpal bones was normal for this age. The blood picture was normal and the Kahn and Kline tests were negative. The blood chemistry studies were interesting.

Feb. 9, 1943	Calcium, 11.9
	Phosphorus, 4.8
	Phosphatase, 72 King units
Feb. 13, 1943	Phosphatase, 63 King units

These phosphatase figures are among the highest ever seen in our laboratory.

Dr. Charles Jacobs, called in orthopedic consultation, applied casts to both legs to correct the contractures. A biopsy taken from the left knee and studied by Dr. I. Davidsohn was reported as follows: Sections showed large masses of calcific deposits staining a deep purplish color. They were surrounded in places by a pink, fairly homogeneous tissue which was infiltrated with large numbers of mononuclear and occasional polynuclear giant cells. The latter had the appearance of foreign body giant cells and were seen engulfing masses of the foreign calcific deposits. The pink tissue had the appearance of synovial membrane. There was no evidence anywhere of new bone formation. We are dealing here with a degenerative process, with deposits of calcium and a reactive inflammation of the synovial membrane. *Diagnosis:* calcification of synovia;

reactive inflammation. *Microscopic report:* many red blood cells; 3 to 5 white blood cells per high power field. *Smear:* no bacteria seen. *Culture:* no growth aerobically nor anaerobically.

Fig. 4.

Some light is thrown on the prognosis of this bone abnormality by Raap's observation of a 2½-year-old female whose three younger brothers revealed these changes. She showed normal bone structures except for a few punctate densities in both wrists and ankles. He considered this as possible evidence that she may previously have had the same

changes. He says "apparently these abnormal calcifications are marked at birth and disappear by the age of about 3 years."

Our patient has progressed satisfactorily in spite of this bone abnormality and the congenital heart lesion. Weight at 8 months was 15 pounds, 7 ounces, and length was 27¾ inches. He has been on a regular diet of evaporated milk formula, cereals, egg yolk, vegetables, des-

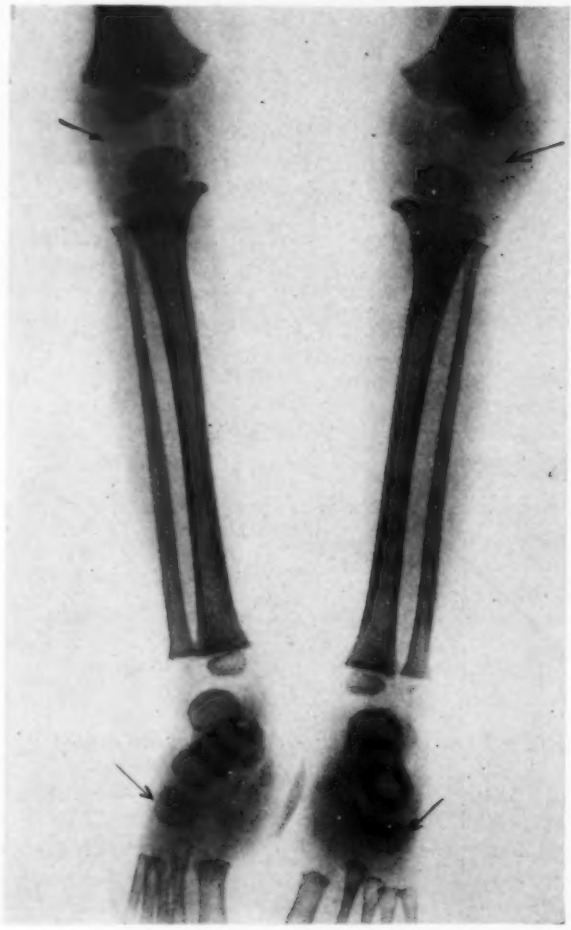

Fig. 7.

serts, orange juice, and percomorph oil (10 drops a day). This latter item was one of considerable concern in view of the extensive calcification seen at the knee joints, but it was thought best to incorporate the usual amount of vitamin D in this infant's diet to insure protection against rickets.

Just what part, if any, was played by the trauma to the knees during the breech extraction in the ultimate tissue change and extensive calcification cannot be determined, but it probably was a localized factor. Our original contention that these shadows were caused by calcified hematomas was not borne out by biopsy.

The x-ray findings have shown no improvement in the condition during our observation for several months, but more normal development is anticipated in a few more years if this case conforms to the course of other previously observed patients. Light casts are still necessary to prevent contractures at both knees.

SUMMARY

1. A case is reported in which there is bone maldevelopment which consists of stippling at many epiphyseal centers.

2. Extensive calcification was noted in both knee joints.

3. Periosteal elevation along the femur was a new finding.

4. An associated abnormality was the congenital heart lesion (also found in two other patients with similar conditions).

5. No evidence of thyroid deficiency was noted, although other observers are of the opinion that hypothyroidism is an associated condition.

6. An extremely high phosphatase determination was discovered.

7. Placing this case in the category of chondrodystrophies seems most logical.

8. Prognosis for correction of the bone abnormality seems generally good.

9. No special treatment seems necessary unless thyroid be given empirically.

AUTHORS' NOTE

Since submission of this paper for publication, x-ray films were taken of the patient at 1 year of age. Figs. 6 and 7 show almost complete absorption of the calcium deposits at the knees and the appearance of normal bone formation in the metatarsal area where stippled bone was previously present.

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MENINGITIS DUE TO SALMONELLA ORANIENBURG

Report of a Case

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MENINGITIS due to the Salmonella group of organisms is rare, not more than fifty-three cases having been reported. We wish to report a case in which the infecting organism was the *S. Oranienburg*. This is the first case to be reported of meningitis resulting from infection with this strain of Salmonella, although one other case has been tabulated as due to this organism.[4]

B. S. L., a 5-week-old male infant, was admitted to the Boston Floating Hospital July 4, 1941, because of convulsions of nine days' duration.

The family history was noncontributory and there was no illness of any kind at the time in any member of the family or household. The prenatal period was uneventful. Birth was normal, occurring three and one-half weeks prematurely; birth weight was 6 pounds, 4 ounces. The neonatal condition was good, marked only by "jaundice of the newborn." He was discharged in good condition at the age of 3 weeks, at which time he had nearly regained his birth weight. He was on a Biolac formula, and since 3 weeks of age had received accessory vitamins. He went home to a dairy farm near Boston and did well up to the onset of the present illness. Nine days prior to admission he began to have twitchings of the mouth, both eyelids, hands, and arms, lasting for a few minutes at a time and occurring perhaps three or four times a day. There were no other complaints—no fever, vomiting, refusal of feedings, or diarrhea. Four days before admission he began to have "spells" of screaming and holding his breath. These episodes lasted five or ten minutes, during which time the patient's body became rigid and his color cyanotic. Following the seizure he was pale and quiet for a short time. Without other symptoms this persisted until the day of admission, when he had five such attacks.

Physical examination at the time of admission showed a 5-week-old male infant of normal development in a good state of nutrition and hydration. The general appearance and behavior showed nothing unusual. The head was symmetrical and showed no evidence of injury. The fontanels were both open and of normal tension. The eardrums showed no inflammation. It was thought that the right optic disc showed some blurring of the outlines; the fundi were otherwise negative. The pharynx was slightly injected. There was no neck rigidity and no cervical lymphadenopathy. The lungs showed slight impairment of the percussion note in the right infraclavicular region but no definite signs of pneumonitis or consolidation. The heart was not enlarged, there were no adventitious sounds, and the cardiac action seemed normal. The abdomen was soft, with no areas of rigidity or apparent tenderness and no unusual masses. The reflexes were all hyperactive. Babinski's and Kernig's signs were negative and there was no spasticity or paralysis of the muscles of the extremities.

The diagnosis was not clear on the day of admission. Blood calcium and phosphorus levels were 11.2 mg. per cent and 6.3 mg. per cent, respectively, and the blood morphology showed a moderate anemia (red blood cells, 3,780,000; hemoglobin,

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This case was included briefly in a report of a series of cases of meningitis.[4]

73 per cent) and a leucocytosis (20,200) with increased neutrophiles (61 per cent). The following day a lumbar puncture yielded turbid fluid with a total cell count of 1,350, of which 95 per cent were neutrophiles. Globulin was increased. Sugar was so low that quantitative determination could not be made. Chlorides were within normal limits (638 mg. per cent). Direct smear of the spinal fluid showed many small, thick, gram-negative bacilli and the same organism grew out on culture. This was thought to be a Salmonella but could not be specifically identified as to variety at the time. Daily lumbar punctures showed this organism on smear and culture consistently for the first week. The spinal fluid was always purulent, the cell count varying from 560 to 1,750 cells per cubic millimeter. In spite of repeated lumbar punctures and sedation the patient continued to have several convulsions each day during the first and second weeks in the hospital. Clinically he showed little change; he ate fairly well and gained one and one-half pounds during the first two weeks. Treatment consisted of sedation, frequent lumbar punctures, and supportive treatment in the form of two transfusions, one of 100 c.c. on the second day and one of 60 c.c. on the eighth day. Sulfathiazole was started on the second day (the day of the first lumbar puncture) in doses amounting to 10.5 gr. daily (about 1½ gr. per pound of the patient's weight). Two days later this was increased to 24.7 gr. daily, which was about 3 to 3½ gr. per pound of body weight. With this dosage the sulfathiazole level in the spinal fluid on the fourth day was 5 mg. per cent and on the twentieth day, 3.8 mg. per cent. Repeated blood cultures were sterile. Toward the end of the second week the patient showed a gradual improvement. The convulsions became less severe and less frequent and finally ceased. His appetite and general condition were better and three consecutive negative spinal fluid cultures were obtained on the tenth, twelfth, and fourteenth days. The improvement was temporary, however, and the cultures became positive again on the sixteenth day. At the same time the dynamics of the spinal fluid together with an apparent change in the clinical condition (drowsiness, staring expression) suggested the presence of a block; this was further confirmed on the twentieth day, when a lumbar puncture gave a negative culture and a ventricular puncture gave a positive culture. The head circumference at that time was 37.5 cm. Thereafter, ventricular punctures were done every day or on alternate days, always yielding purulent fluid and positive culture. The ventricular fluid became increasingly thick and purulent; on one occasion the cell count was 9,500 cells per cubic millimeter. The course from the beginning of the second month was gradually downhill. The patient began to have slight jerky convulsions; he slept most of the time; he began to refuse his feedings and later to vomit. His head circumference increased rapidly; twenty-nine days after the first measurement it had grown to 45 cm., an increase of 7.5 cm. He became more and more listless and finally expired on the fifty-fourth hospital day.

*Autopsy.**—The examination of the body and extremities may be summarized as follows: No congenital anomalies were found except for a small inguinal hernia. The heart was of average size and weight and showed no pathologic changes. The lungs were soft and crepitant without exudate or consolidation. The trachea and bronchi were clear. The duodenum and ileum were distended. There was no evidence of enteritis, remote or recent.

The head was unusually large (measured 43 cm. at the autopsy table). The suture lines and fontanels were widely separated and bulging. There were extensive adhesions between the brain and the overlying dura, particularly in the left frontal region, in the middle cranial fossa, and about the foramen magnum. The brain was bulging and edematous; the convolutions were flattened and the gyri were practically obliterated. There was a marked medullary conus. There was much thin seropurulent fluid on the surface of the brain, both beneath the dura and beneath the arachnoid. The brain itself was large (weight, 895 Gm. against a normal of 500 Gm.

*Performed by Dr. H. E. MacMahon and Dr. H. E. Olken.

for this age) and almost semifluid in consistency. The lateral ventricles were markedly dilated. They contained fluid which on the left was thin and serous and on the right, thick, greenish, and purulent. Much of the fluid was walled off into small pockets by thin fibrous adhesions. The dilatation of the lateral ventricles was so marked that the surrounding cortex and other structures were greatly compressed and flattened. The interventricular foramen seemed to be sealed off and it was not possible to identify the third ventricle. The fourth ventricle was likewise almost obliterated by dense adhesions between its floor and the cerebellum and the cerebellum contained a small pocket of frank pus. Microscopic sections showed thickening and, in some areas, fusion of the meninges and adhesion of them to the brain substance. Varying degrees of chronic and acute suppurative inflammatory exudate were found within the meninges in walled-off abscesses. The third ventricle was overgrown and obliterated by glial and connective tissue. In the brain substance itself there was perivascular inflammatory exudate and, in some areas, degeneration of nerve cells.

The etiology in this case was definitely established through the kindness of Dr. Philip R. Edwards, of the Department of Animal Pathology, Lexington, Ky. A sample culture was examined by him and the organism was reported to be *S. Oranienburg*. This is a member of the paratyphoid C, or choleraesuis, group of organisms. It is found usually in fowl, and when it occurs in man, which is unusual, it is almost invariably the cause of gastroenteritis, or "food-poisoning."[2, 3, 4] Edwards has found only one other instance in which this organism has been the cause of meningitis.[4] One may speculate on how this organism invaded the infant. Most of the cases of Salmonella meningitis in the literature seem to represent complication of an already existing paratyphoid fever, or, at least, from the symptoms and bacteriologic evidence one may adduce that the portal of entry was via the gastrointestinal tract. A few cases appear to be "primary" meningitis. Whether the present case falls into this group cannot be known, because the stools were not examined bacteriologically; all that can be said is that clinically and at autopsy the infant showed no evidence of enteritis. It is again speculative whether the patient was infected from one of the animals or people on the farm where he lived. It is possible, but careful cultures taken from these sources failed to produce *S. Oranienburg*. Edwards mentions "a number of instances in which (Salmonella) infection in man was traceable directly to animals."

Clinically, there was a moderately acute onset, not the acute fulminating picture seen in the patients of Fothergill and Sweet[5] and Bahrenburg and Ecker,[6] in whom death occurred within twenty-four hours after the onset of symptoms. The blood picture was not remarkable; there was a mild anemia and the white blood cell count ranged between 11,000 and 20,000 cells per cubic millimeter, showing no extremes of variation despite the sustained large doses of sulfathiazole. The spinal fluid showed a marked neutrophilic reaction which agrees with the findings in some of the reported cases. The choice of sulfonamide is open to comment. Barrett and associates[7] recommend sulfa-

thiazole in the treatment of *Bacillus coli* meningitis in infants, reporting two cures, and Hollis and Baty recently reported a similar recovery.[1] On the other hand, sulfathiazole has in general been avoided in the treatment of meningitis because of its relatively low concentration in the spinal fluid. Of the five reported recoveries from Salmonella meningitis with sulfonamides, none of the patients have had sulfathiazole alone. Katsampes and Bradford[8] reported the recovery of an infant with meningitis due to *Salmonella Panama* treated with sulfapyridine. Patterson[9] had two recoveries in patients with paratyphoid B meningitis, one infant and one 8-year-old child, each of whom was treated with both sulfapyridine and sulfathiazole. Wilsdon and Gibson's patient,[10] a 61-year-old man with chronic paratyphoid fever and meningitis as a complication, failed to recover after treatment with sulfapyridine, while Storey's patient,[11] 23 years old, with paratyphoid B meningitis complicating paratyphoid fever, recovered without any chemotherapy. Another recovery was recorded by Valledor and associates[12]—a 3-month-old infant with an acute meningitis due to *Salmonella Eastbourne*. High doses of sulfadiazine were given, achieving a level in the spinal fluid of 10 mg. per cent, and intensive supportive treatment was maintained. After a long illness and a relapse the patient finally recovered but had as a sequela some residual muscular paralysis and apparent mental retardation. Recently, Lyon and Folsom[13] reported the recovery of a child 2 years old with meningitis due to *Salmonella suipestifer*. The patient had a stormy course, recovering only after intensive treatment. The blood sulfanilamide level reached 22 mg. per 100 c.c., and the spinal canal was repeatedly irrigated with sulfanilamide solution. Block was suspected but did not develop and cisternal irrigations with sulfanilamide were done.

Pathologically, the case is similar to the one reported by Gordon and Kennedy,[14] in which the pathologic findings were described in detail. They emphasized particularly the extraordinary inflammatory response in the meninges and the glial proliferation which had taken place during only eleven days of illness. This was probably comparable to the marked gliosis, which entirely obliterated the third ventricle, observed in our patient. Both patients also showed perivascular infiltration in the brain substance itself. Gordon and Kennedy's patient probably had a "primary" meningitis; there was no enteritis, and the infant, who was only 17 days old at the time of onset, had had no possible exposure. On this basis these authors suggested that the case might represent an intrauterine infection, though the mother had shown no signs of disease. There may be healthy carriers,[4] and in this connection Schmidt[15] convincingly showed that his patient, a 6-week-old infant, had derived the infection via the mother's milk. The mother was shown to have the same organism (paratyphoid B) in the stools, though she was clinically free of any evidence of infection.

The first study of cases of Salmonella meningitis was published by Lynch and Shelburne[16] in 1930. They found fifteen cases in the literature at that time and added one case of their own. These cases were all included in a review of the literature in 1938 by Bahrenburg and Ecker,[6] who analyzed thirty-five cases from the literature between 1908 and 1935, including one case of their own, which proved fatal. In this whole group they found only three recoveries and only one among the twenty cases which had been studied closely enough to allow classification of the organism. In all three of the recovered patients the meningeal infection represented a complication of an already existing paratyphoid fever. One recovery occurred in an infant. From this series they concluded that "Salmonella meningitis must . . . be considered fatal until more evidence to the contrary is produced." Kutscher,[17] in the same year, in a study of gram-negative bacillus meningitis, re-emphasized the fatality of Salmonella meningitis, and Facatselli[18] commented that with the present (1939) means of treatment, purulent paratyphoid meningitis was hopeless. These studies did not include two cases of "metastatic" meningitis in children with enteritis, both fatal, mentioned by Ghon in 1907[19] and quoted by Schmidt.[15] Likewise they did not include a case, also fatal, reported by Fothergill and Sweet[5] in 1933, perhaps because the agglutination reactions in this case were inconclusive. Two more cases were published in 1938—that of Storey[11] which survived, and the clinicopathologic discussion of Gordon and Kennedy.[14] Katsampes and Bradford[8] added another case of an infant who recovered from meningitis due to *S. Panama*, and Patterson[9] had two patients with meningitis due to *Salmonella schottmülleri*, both of whom recovered (mentioned before). In 1937 Ravitch and Washington[20] published a case of mixed infection, meningococcic and suipestifer septicemia and meningitis. The two aforementioned cases of Wilsdon and Gibson[10] (*Salmonella London*) and Lyon and Folsom[13] (*S. suipestifer*) finish the list of American, British, and German literature. Between 1938 and 1942 six cases were reported from Latin America. Santiago[21] had three cases of fulminating meningitis due to *Salmonella aertrycke* in newborn infants, all of whom died. Caselli and Teobaldo[22] described a fatal case of Paratyphoid B meningitis in an infant, complicating a *B. coli* cystitis and a *Bacillus typhosi* blood stream infection. Peluffo and associates[23] discussed a fatal case of meningitis due to *Salmonella typhi murium* in an infant who also had tuberculous mesenteric glands, and they rejected the possibility of tuberculous meningitis in the patient. Valledor and associates[12] reported a recovery from *S. Eastbourne* meningitis (mentioned before).

Adding all these cases together, with the exception of the mixed case of Ravitch and Washington, there appear to have been fifty-three cases of Salmonella meningitis reported in the literature, including the present case and the other case of *S. Oranienburg* meningitis mentioned by

Edwards. There have been apparently not more than nine recoveries: three in Bahrenburg's series, one reported by Storey, one by Katsampes and Bradford, two by Patterson, one by Valledor, and one by Lyon and Folsom. Of the nine patients who recovered, sulfonamide therapy was used in five; four of the five were infants. Of the forty-four patients who died, sulfonamides were used in only two: Wilsdon and Gibson's (sulfapyridine), and ours (sulfathiazole). In other words, of the seven patients who received sulfonamide therapy, five survived, four making a complete recovery, while of the forty-six patients who did not receive sulfonamide therapy, only four recovered.

SUMMARY

A fatal case of meningitis in an infant due to *S. Oranienburg* is reported, together with the important autopsy findings.

The literature on Salmonella meningitis is reviewed briefly.

The results suggest that sulfonamide therapy is effective in certain types of Salmonella meningitis. The best results have been obtained with sulfapyridine (three cures) and sulfanilamide (one cure).

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BEZOAR CAUSING INTESTINAL OBSTRUCTION

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TRICHOBEZOAR, or hairball, is a rare cause of intestinal obstruction, although hair tumors in the stomach are not so uncommon. The chewing and swallowing of hair, trichophagy, is a habit confined almost entirely to girls. Contrary to general belief, insanity can rarely be indicted in the causation of the habit. In 1915 Matas† made an excellent review of the literature, collecting reported cases of seventy-six children with gastrointestinal hairballs, of whom forty-seven were operated upon, with two deaths. Both of the deaths followed enterotomies in children suffering from intestinal obstruction. Most of the operations were gastrotomies, and in only ten patients had a correct preoperative diagnosis been made. Usually the masses were palpable, movable, but not tender, and sometimes weighed over two pounds. The following case is of interest because of the past history and laboratory findings.

R. L., a 13-year-old girl living in Torrington, Wyo., was admitted to Children's Hospital, Denver, Nov. 11, 1942, because of extreme emaciation, vomiting, and abdominal pain. Both parents and nine siblings were living and well. The patient was born prematurely, weighing 3¼ pounds (1.47 kg.). Her development had been quite normal. Chicken pox, mumps, and scarlet fever in early childhood were not accompanied by any complications. A history of pica and of pulling out and of chewing her hair in early childhood was not obtained until after death.

At the age of 10 years, or three years before admission, appendicitis had been diagnosed. Following the removal of the appendix, the patient was seriously ill for six months. Vomiting, distention, abdominal pain, and extreme emaciation accompanied this illness. Without further surgical intervention these symptoms finally disappeared; the child rapidly gained weight and was in excellent health until six weeks before admission. At this time, the same complaints returned suddenly, and, one week after the onset, a laparotomy was done because of a palpable mass in the right side of the abdomen. However, the mass was not located at the operation and the signs of intestinal obstruction persisted; her critical condition prompted removal to Children's Hospital, Denver.

On admission, the patient was in extremis. Weight was recorded as only 50 pounds (22.7 kg.). The skin was dry and sallow, and the tissue turgor was poor. Purpuric spots were present in both antecubital fossae. The ribs and bony prominences testified to an extreme state of emaciation. Bedsores were present in the sacral region. The tongue was dry and furrowed, and the mucosa of the soft palate and pharynx presented an orange-red appearance. Fetor exoris was noted and the patient belched frequently. The skin of the abdomen was discolored, reportedly from a hot water bottle burn. Two scars were present in the right lower quadrant, one old and one healing. The abdomen was so distended that palpation was difficult, but a firm mass, not tender and apparently movable, was found in the right flank. At times the mass was ballotable. Other positive find-

*Dr. Forbes died Nov. 8, 1943, a few days after this manuscript was received.

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†Matas, Rudolph: Surg., Gynec. & Obst. 21: 594, 1915.

ings included absent tendon reflexes, some pallor of the optic discs, and profound muscular weakness. Examination of the chest revealed no significant findings.

The temperature on admission was 100.2° F. (37.9° C.) but rose to 103° F. (39.4° C.) before death on the following day. The pulse rate was 120 per minute and of poor quality and the blood pressure was 58/18. Laboratory findings showed a severe disturbance in the metabolism. Hemoglobin was 58 per cent. Red cells numbered 3,200,000 and leucocytes, 17,900, of which 91 per cent were polymorphonuclears. Toxic granulation was marked in the stained smears. The bleeding

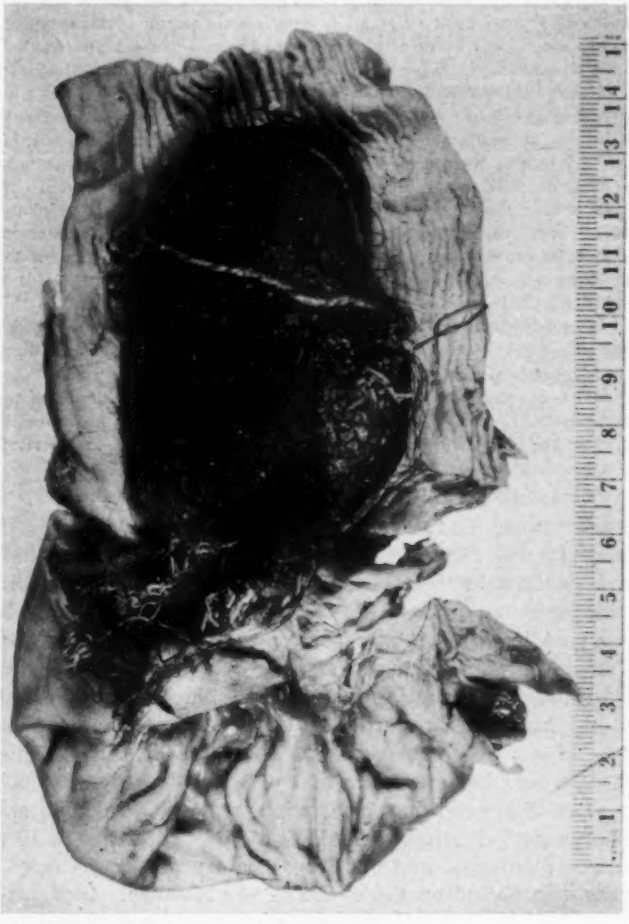

Fig. 1.

time was five minutes, forty-five seconds. Platelets numbered 158,000. Serologic tests for syphilis were negative. The nonprotein nitrogen was 31 mg. per cent and the serum total protein, 4.5 per cent. Blood cholesterol was 188 mg. per cent and fasting blood sugar, 103. The carbon dioxide combining power of plasma was 59 volumes per cent. The most alarming evidence of impending dissolution was the hypochloremia and low serum sodium. Serum chloride was found to be only

342 mg. per cent; the serum sodium, 257.5 mg. per cent; and the potassium 13 mg. per cent. The urine was scanty and dark and contained sulfa crystals.

Treatment was directed at restoration of fluids and chloride. Normal saline, 1,000 c.c., was given intravenously followed by 250 c.c. of whole blood. Eschatin, 5 c.c., was injected and the blood pressure rose to 96/24. Nausea, vomiting, and abdominal distention continued and restlessness increased; the patient died twenty-four hours after admission. Autopsy was performed by Dr. H. D. Palmer.

Autopsy Protocol.—Subcutaneous fat was practically absent. There was extreme distention of the small intestine throughout its entire length. In the lower right quadrant, several coils of small intestine were purple-black in color. The omentum was spread out anteriorly over the abdominal content and was adherent to the operative scars. A mass was located within the lumen of the ileum at a distance of about three feet from the ileocecal valve. Volvulus of the mesentery had occurred in the lower portion. One complete turn was found. The wall of the intestine in the portion so obstructed was the seat of hemorrhagic infarction. The loops of intestine were sealed together by fibrin and the surfaces were dull. There were some fibrous adhesions in the operative area. The small intestine varied from 8 cm. in diameter in the lower portion to 12 cm. in the jejunum and duodenum. There was at least 5 L. of intestinal content. The stomach was dilated about 50 per cent and contained some fecal matter. The mass found in the lower ileum measured 9 by 5 by 5 cm. (Fig. 1). Dissection proved it to be a bezoar containing hair, string, and thread. No hairballs were found in the stomach. The adrenal glands presented no destructive changes and no evidence of diffuse atrophy. The changes in the other organs were those of prolonged malnutrition, dehydration, exsanguination, and toxemia.

In summary, a bezoar in the terminal ileum must be considered the essential pathology. It started the train of events which led to the patient's death. The final events in the chain were volvulus, infarction of the bowel, and peritonitis.

COMMENT

After the autopsy findings were reported, a letter was received from the mother stating that the patient "used to pull out her hair and chew and swallow it." From both the pediatric and surgical viewpoint, this unfortunate case illustrates the value of a complete and searching past history. It also portrays the extreme degree of electrolite imbalance that may occur as the result of vomiting alone. The hypochloremia, presenting the lowest serum chloride thus far encountered in our hospital, can hardly be explained except on the basis of vomiting. If one were to exclude the abdominal findings in this case, the picture would be essentially that of adrenal cortex insufficiency; that is, arterial hypotension, emaciation, extreme weakness, and low serum sodium and chloride. The first episode (at the age of 10 years) of vomiting, distention, and abdominal pain probably occurred when the bezoar was occluding the outlet of the stomach. Obstruction at that time must have been nearly complete, since the mother stated that emaciation was just as marked then as during the second fatal episode. The three-year asymptomatic interval between attacks is difficult to explain, but it is plausible to suggest that the foreign body may have moved back into the stomach at the time of recovery from the first episode and that the final illness was coincident with its passage from the stomach to the place of its final lodgment in the ileum.

STAPHYLOCOCCUS EMPYEMA IN INFANTS AND CHILDREN

A Review of Twenty-Nine Cases

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WITH the advent of sulfonamide therapy the incidence of empyema in all age groups has fallen off in striking fashion. With this the occurrence of empyema caused by organisms unaffected by the drug has remained approximately constant and consequently has shown a marked relative rise. As a result staphylococcus empyema has become proportionally more important. Of the vast literature on suppurative pleurisy, only a small portion is devoted exclusively to that caused by this organism and, as the outlook for these patients has always been considered grave, it was thought worth while to review the experience with staphylococcus empyema at Babies Hospital.

Material.—The cases studied here were collected from the Babies Hospital records over a period extending from 1922 through the early part of 1943. During the first seven years of this twenty-one-year span the upper age limit of patients admitted to Babies Hospital was 4 years; in the latter two-thirds of the period children up to 13 years of age were accepted. This change of policy had less effect on the incidence of cases of staphylococcus empyema than might at first be supposed, for among the total number of cases covered by this report, there was only one occurring in a patient more than 2½ years of age. The results of treatment should be evaluated with due consideration of the fact that an appreciable turnover of staff was unavoidable in view of the time period covered by this survey.

Incidence of Staphylococcus aureus as the Causative Organism.—In Fig. 1 the incidence of staphylococcus infection in empyema relative to other agents is shown graphically. This represents the experience at Babies Hospital over an eleven-year period from 1932 through 1942. It will be seen that through 1937 (prior to the extensive use of sulfonamides) pneumococcus predominated strikingly and fell off thereafter. In terms of percentage staphylococcus as the causative agent represented 7.3 per cent of all cases of empyema in the first period and 20.4 per cent in the last five years. Despite this gain in relative frequency the actual incidence of the disease has remained about constant, so that sulfonamides have apparently had little effect on it. Since a decrease of this sort in the frequency of pneumococcus empyema and consequently of empyema in general would be expected with the introduction of sulfapyridine

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The opinions and assertions contained in this article are the private ones of the writer and in no way reflect the opinions of the Navy Department.

(1938), it is surprising that in a recent report by Berman[1] no significant change in the frequency of cases of empyema was observed.

Figures taken from the reports of various authors, depicting for the most part the experience of the presulfonamide era (Table I), are in approximate agreement with the first figure cited in the preceding paragraph. Totaling the cases in the whole pediatric age group reported by the authors noted in Table I and calculating the percentage of cases

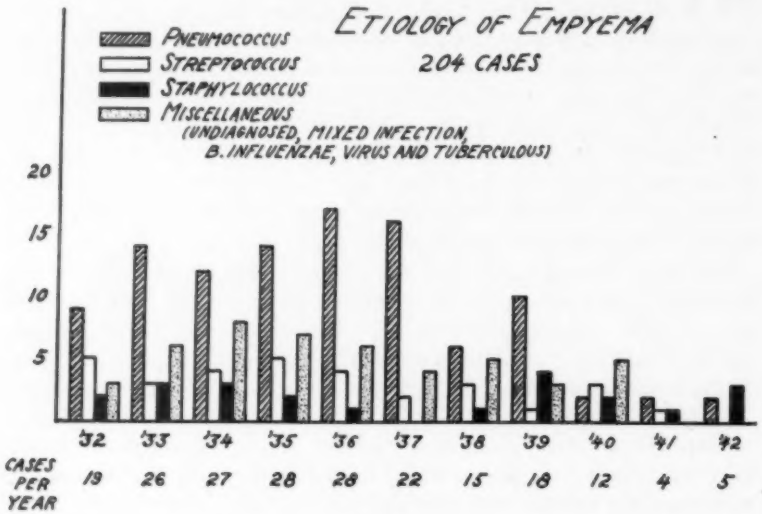

Fig. 1.

TABLE I

INCIDENCE OF STAPHYLOCOCCUS AS THE CAUSE OF EMPYEMA, AS ASSEMBLED FROM REPORTS IN THE LITERATURE

AUTHOR	DATE	NO. OF CASES TOTAL	STAPH. CASES	PER CENT
Holt and Howland[6]	1913	180	14	7.4
Rienhoff and Davison[13] (under 2 yr.)	1928	(*41*)	(*8*)	19.5
Hart[3]	1930	36	7	19.5
McEnery and Brennemann[9]	1928-1932	168	14	8.3
Neuhof and Berck[10]	1928-1932	373	42	11.3
Hochberg and Kramer[4]	1929-1936	300	28	9.3
Babies Hospital	1932-1937	150	11	7.3
Lanman and Heyl[8]	1919-1938	467	42	9.0
Nowak[11] (adults included)	1939	(*500*)	(*38*)	7.6
Hochberg and Steiner[5] (under 6 mo.)	1940	(*51*)	(*19*)	61.3
Penberthy and Benson[12]	1926-1941	144	9	6.2
Ellenberg and Martin[2]	1942	96	8	8.3
Total		1,914	175	9.1
Babies Hospital	1938-1942	54	11	20.4

Reports in italics omitted in total because they were not representative of whole pediatric age group.

of empyema caused by staphylococcus through 1941, a frequency of 9.2 per cent is derived. It will be noted that the proportion of cases of staphylococcus empyema in surveys limited to the first two years of life is considerably higher than in the others.

Age Incidence.—All but one of the patients in our series were less than 2½ years of age. The exceptional case, in a patient 4 years of age, was furthermore unusual in that it was associated with a severe agranulocytosis. The preference of staphylococcus empyema for the younger age group is strikingly demonstrated in Fig. 2. Staphylococcus empyema is thus seen to be a disease which most often attacks

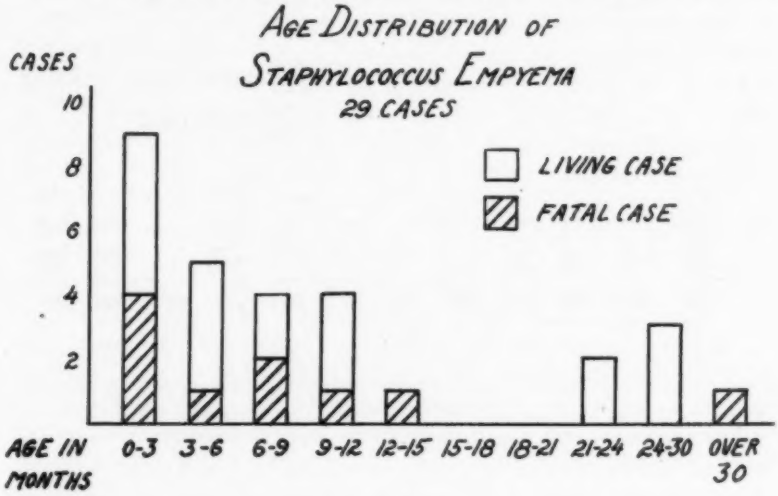

Fig. 2.

infants less than 1 year of age. Of the twenty-nine cases, twenty-two (75.9 per cent) were in patients less than 1 year of age, and of these, nine (40.9 per cent) occurred in the first three months of life. This experience shows a higher incidence in the infant age group than that of Neuhof and Berek,[10] who report 42 per cent of thirty-one cases in the first year, and even higher than that of McEnery and Brennemann[9] who had 66.6 per cent of nine cases in the first year.

Seasonal Incidence.—Fig. 3 shows the occurrence of staphylococcus empyema classified by months of onset. It is interesting to observe that there is no striking variation by season, although in this series March proved to be the peak month. There are, however, an appreciable number of cases in the summer months when respiratory infection should be at its lowest in this climate. It would seem from this admittedly limited series that prevalence of respiratory infection is not as significant as is the circumstance that the host is in the very low age group where he is not capable of combatting infection when exposed.

Primary Disease.—It has been said that staphylococcus empyema, as well as other types of empyema, is rarely, if ever, primary.[5, 9] The experience in this group of cases would tend to bear that out. Twenty-three of the twenty-nine patients had what at least clinically seems to have been pneumonia prior to the onset of the pleural effusion. In this connection it is interesting that Kanof, Kramer, and Carnes,[7] in studying thirty-seven cases of staphylococcus pneumonia, found empyema in twenty-five. Two patients, one a newborn infant and one 7 weeks of age, had what was apparently generalized sepsis (one shown with a

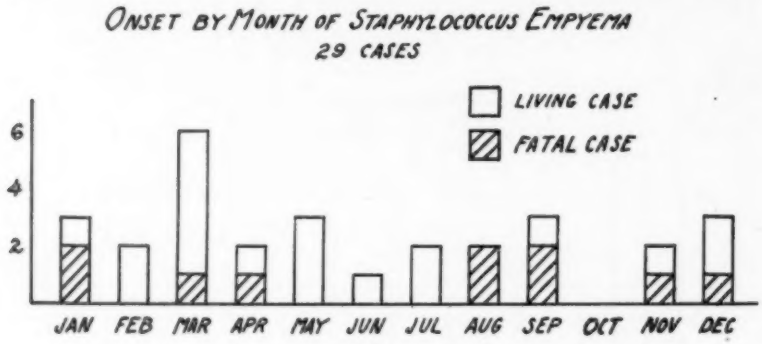

Fig. 3.

positive blood culture), in which the empyema was demonstrated only at autopsy. Of the remaining four cases, one patient had chylothorax which apparently became secondarily infected; another, 4 years old, had agranulocytosis and septicemia and showed a mixed infection of the empyema fluid with *Streptococcus hemolyticus* as well as with *Staph. aureus.* One had a loculated empyema, apparently of long standing, discovered incidentally in the course of a hospital admission for intussusception. Finally, there was one patient in whom the history of illness, before the presence of a large amount of fluid was detected, was so brief that the empyema could conceivably have been truly primary.

Blood cultures were taken in all but five of the twenty cases since 1932 and found negative in all but the one instance mentioned in the preceding paragraph. Therefore bacteremia was not a feature by the time the patients were admitted to the hospital. It is possible that this is explained by the fact that patients capable of developing empyema have sufficient resistance to avoid bacteremia. Mixed infection was noted in four of the patients: in the patient with fatal agranulocytosis mentioned earlier; in a 19-day-old infant who showed *Hemophilus influenzae* associated with *Staph. aureus* at the first pleural aspiration, but whose empyema fluid later contained a pure culture of *Staph. aureus;* in a 7-month-old child who developed bronchopneumonia and suppurative pleurisy following a tracheotomy, whose empyema fluid contained

Str. hemolyticus associated with staphylococcus; and in a 23-month-old infant in whom *Str. hemolyticus* was present. All three of the last-mentioned patients recovered.

Treatment.—In the earlier cases no specific medical therapy was attempted—only general supportive treatment, including transfusions where indicated. More recently various types of medical therapy hoped to be specific for the organism have been used. Nine patients received sulfonamides; in this group there were three deaths. Four patients were given bacteriophage parenterally, locally, or both; two deaths resulted. Two were treated with staphylococcus antitoxin; one death resulted. These individual agents were tried both alone and in combination with each other, and in none of the patients was there anything in the clinical course to suggest a truly beneficial result.

With these disappointing medical results, it is apparent that the only satisfactory approach is one in which attention is directed to removal of the pus. Such an approach is here considered surgical, although in some instances only thoracentesis was used. In general, no predetermined course was followed, but an effort was made to evaluate each case separately. When the diagnosis of empyema was suspected, a diagnostic aspiration of the chest was performed, removing as much fluid as possible at the time. Subsequently further aspirations were done as fluid accumulated and as they seemed necessary. If in the course of the next week or two weeks the fluid still tended to reaccumulate, a thoracotomy was done with intercostal drainage, if the lung and mediastinum did not seem fixed, or rib resection if they did. This procedure was not followed in every case, since in a few "siphon drainage" was instituted immediately after diagnosis. Table II shows the figures for the various forms of treatment.

TABLE II

	PATIENTS UNDER 1 YEAR		PATIENTS OVER 1 YEAR	
	CASES	DEATHS	CASES	DEATHS
Thoracentesis alone	9	5	1	1
Intercostal drainage	4	0	2	1
Rib resection	7	1	4	0
No therapy (diagnosed at autopsy)	2	2	–	–
Total	22	8 (36.4%)	7	2 (28.6%)

The high mortality shown for thoracentesis alone results in part from the fact that a good many of the babies in whom more extensive surgery should have been done were moribund or at least too sick for such treatment. But the fact that four patients recovered with needle aspiration alone shows that open thoracotomy is not always necessary. The time these babies spent in the hospital after diagnosis varied from twenty-nine to fifty-four days. The babies who died did so in from five to sixteen days.

Those children who recovered with intercostal drainage remained in the hospital varying lengths of time, from twenty days in two instances to seventy-seven days in one. In the latter instance the child had a mixed infection with *H. influenzae*. The eventual outcome in all these patients was satisfactory.

The patients who had rib resection were those who had proved refractory to more conservative treatment. In five of these, intercostal drainage had proved inadequate, and in the others, only thoracentesis had been tried. The shortest time after the diagnosis in which rib resection was done was seven days; the average was 18.4 days and the longest was thirty-two days. Hospitalization following the procedure varied from twelve to 145 days, with no obvious relationships to type of therapy demonstrable. As far as could be determined, all patients eventually recovered completely, although in two of them about two years were required for all drainage from the thoracotomy wound to cease; one of these was the child with chylothorax mentioned previously. In those few instances in which scoliosis was noted, it had either cleared completely at discharge or had shown progressive diminution in severity.

Complications.—Complications attributable to therapeutic attempts were not frequent. Chest wall abscesses following needle aspiration were noted in six patients and osteomyelitis of the ribs was seen twice following rib resection. Bronchopleural fistula occurred only once.

Mortality.—The distribution of deaths by age, season, and type of treatment has already been shown (Figs. 2 and 3 and Table II). The gross mortality for the whole series was ten cases (34.5 per cent). When an arbitrary division of the entire experience is made, with the winter of 1931-1932 as dividing line, it is found that the mortality rate from 1922 to 1931 is five out of nine (55.5 per cent); from 1932 to 1943, only five out of twenty (25 per cent). This apparent improvement in results is of borderline statistical significance but may perhaps reflect some favorable progress in general therapeutic policies and practices. Our mortality rates are of the same general order of magnitude as those reported in the literature (Table III).

TABLE III

OBSERVER	YEAR	CASES	DEATHS	PER CENT
Rienhoff and Davison[13] (under 2 yr.)	1928	8	3	37.5
McEnery and Brennemann[9]	1932	14	6	42.8
Lanman and Heyl[8]	1938	42	4-5	11.0
Hochberg and Steiner[5] (under 6 mo.)	1940	11	6	54.5
Nowak[11]	1941	38	16	42.0
Total		113	36	31.9
Babies Hospital	1922-1931	9	5	55.5
Babies Hospital	1932-1942	20	5	25.0
Total		29	10	34.5

The mortality figures in Table III are considerably higher than most of the reported figures for the mortality rate of empyema caused by all organisms but are somewhat lower than the death rate reported as occurring in patients in the first year of life. These high figures do not necessarily imply that *Staph. aureus* is more lethal than other organisms, but they can be explained by the age group in which the disease tends to occur.

The cause of death in three patients seemed to be the widespread underlying pneumonia. In two patients empyema was an incidental finding in deaths which clinically appeared to be due to sepsis. One death followed rib resection, and this and the four remaining deaths appeared clinically to be the result of overwhelming infection, although blood culture was positive in only one patient. Death in each instance occurred in from five to twenty-two days after diagnosis, with individual cases being scattered evenly between the limits. In no instance could the empyema alone be called the cause of death. In only one patient who died had the infection apparently become sufficiently localized to warrant rib resection; following this operation the baby appeared to be doing so well that he was sent home, where death occurred unexpectedly with no details known.

SUMMARY AND CONCLUSIONS

The experience at Babies Hospital in New York with a series of twenty-nine cases of empyema thoracis caused by staphylococcus infection is presented and compared with other reports in the literature.

1. The incidence of empyema caused by all organisms has fallen off markedly since the introduction of sulfonamide therapy. Despite this, that caused by *Staph. aureus* has remained about constant, and consequently has increased in relative frequency.

2. About 76 per cent of the cases occurred in the first year of life, with the bulk of these in the first three months.

3. There was no striking seasonal incidence, although there was a slight peak in March. From this it was suggested, because of the age group, that the lack of host resistance rather than the prevalence of respiratory infection was the important factor.

4. About 80 per cent of the cases were preceded by at least clinical pneumonia. Of the remaining, all but one patient had underlying primary disease; this one case might conceivably have been "primary empyema."

5. Various forms of medical treatment were tried; except for general supportive therapy, none seemed to have any demonstrable beneficial effect.

Surgical therapy followed no predetermined policy, except that open thoracotomy was delayed until more conservative treatment proved unsuccessful and until there was reason to believe that the lung and

mediastinum were fixed. Four patients recovered with thoracentesis alone, five recovered with intercostal drainage only in addition to thoracenteses, and ten recovered completely following rib resection. Hospitalization time varied with the individual patient, but, in general, those in whom only thoracentesis was necessary were discharged earlier. From this it was concluded that the surgical approach has to be determined by the response of the patient to his infection rather than by any predetermined rule.

6. Complications following therapy were not a serious problem; abscess at the site of needle puncture was the most common, but osteomyelitis following rib resection occurred twice.

7. The gross mortality for this series was 34.5 per cent, which is in fair agreement with the gross mortality among 113 cases reported in the literature where the death rate was 31.9 per cent. Widespread pneumonia and overwhelming generalized infection seemed to be the causes of death and in no case could the death be attributed directly to the empyema.

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Erratum

In the article by Hardwicke, "Studies on the Minimal Effective Dose of a Water-Soluble Vitamin K. Substitute in the Prevention of Hypoprothrombinemia in the Newborn Infant," which appeared on page 259 of the March issue of the JOURNAL, the following corrections should be noted:

p. 261—The sentence beginning on the twelfth line from the bottom should read: "Twenty-five hundredths of a cubic centimeter, or five drops of the full-strength solution (equivalent to 1.25 mg.), was given once to each of six babies. . . ."

p. 266—On the first line the dose should read 1.25 mg. instead of 0.5 mg.

American Academy of Pediatrics

Proceedings

TWELFTH ANNUAL MEETING OF THE AMERICAN ACADEMY OF PEDIATRICS

CHICAGO, ILL., NOV. 5-7, 1942

Round Table Discussion on Psychologic Management of Infants and Mental Health Problems in Children

Chairmen: Bert I. Beverly, M.D., Chicago, Ill.
Horton Casparis, M.D.,† Nashville, Tenn.

Associates: C. Anderson Aldrich, M.D., Chicago, Ill.
John C. Montgomery, M.D., Detroit, Mich.
Lee Forrest Hill, M.D., Des Moines, Iowa
Clifford D. Sweet, M.D., Oakland, Calif.

Secretaries: Sterling H. Ashmun, M.D., Dayton, Ohio
John Vonachen, M.D., Peoria, Ill.
Norman T. Welford, M.D., La Grange, Ill.
John F. Sander, M.D., Lansing, Mich.

Developmental Perspective in Child Management

Norman T. Welford, M.D., La Grange, Ill.

Discussion by Arnold Gesell, New Haven, Conn.

The intelligent protection of the mental health of the young child presupposes a useful theory of the mind and a philosophy of development. From a medical standpoint, we may think of the mind as a living "structure" which is constituted of a changing complex of behavior patterns. Like the body, the mind has shape, has form. Indeed, we need to make no undue distinction between mind and body.

The infant is a growing action system. He comes into possession of his so-called mind in the same way that he comes into possession of his physique; namely, through processes of growth. As the nervous system of the child matures, the forms of behavior take shape in lawful and orderly sequence. Mental development, therefore, yields to diagnosis. Development also yields to well-conceived guidance measures.

Development, as well as disease, falls within the scope of clinical pediatrics. To effect an adequate supervision of mental growth, we must recognize the importance of the factors of age and maturity in shaping the behavior of the child. We cannot solve the practical problems of mental health guidance in terms of absolute and rigid discipline and habit formation. We must think in relative terms: We must envisage the ever-changing problems of development in their progressive perspective. Habits are fixed modes of behavior; the child is not fixed. He grows, and as he

†Deceased.

grows his "habits" change. In fact, growth is to no small extent a process of rehabituation. This principle has far-reaching consequences. It means that we must approach the problems of child management in terms of their relativity. We must envisage the behavior of the child in its developmental perspective.

Development does not proceed in a straight line; it deviates now up, now down, now left, now right. Sometimes it even seems to deviate now backward, now forward, but the total trend is forward. If the deviations are not too many and too extreme, the organism catches its balance at each step and then makes another step onward. The fluctuations are really not lapses—they are groping "efforts" of the organism to reach a further organization.

Parents are much too prone to think of the problems of child care in terms of discipline, training, and habit formation. This leads them astray. They should think not in absolute concepts, but in the relative concepts of growth. Growth is the key concept. The pediatrician can do a great deal to impart the developmental point of view.

We cannot fully protect the mental health of the infant; we cannot make democracy a genuine folkway unless we bring into the homes of the people a developmental philosophy of child care—an informed philosophy which thinks of *growth* as a concrete, living process rather than a mere label. The growth of the mind is a series of actual events governed by laws and forces just as real as those which apply to an internal combustion engine. But an engine is more easily understood than the child, because the child is constantly passing through stages of developmental metamorphosis. His mind is a growing organism, and whenever it makes a forward thrust at its growing margin, it produces new patterns of behavior.

During this thrusting period the organism is in a state of relative instability. The innovations are then integrated throughout the total action system. There then follows a period of relative equilibrium, which, in turn, is followed by another forward thrust. As the child matures, he progresses through more or less well-defined periods of innovations-integration-equilibrium, innovations-integration-equilibrium, etc.

The 4-week-old child is in more stable equilibrium than the newborn infant. The 8-week-old child is undergoing changes which produce transient manifestations of disequilibrium. At 16 weeks he is again in relative equilibrium. This is followed by a transitional period in which he shows a dawning sensitiveness to strangers. He is not quite equal to combining his self-activity with social behavior. But at 28 weeks he is more composed.

The typical 28-week-old infant presents a classic picture of balanced self-containment, sociability, and general contentment, whether in the supine, prone, or seated position. But this period of amiable equilibrium dissolves developmentally into another period of transient disequilibrium. This is the general formula of development throughout the period of infancy and childhood. There are periods of recurrent equilibrium at 1 year, 18 months, 2, 3, 4, and 5 years. These periods are not sharply defined, but they are offset by periods of relative readjustment and reorganization which tend to become more prominent during the intermediate ages.

The 2½-year-old is in such a period of reorganization. He furnishes an excellent illustration of the mechanisms of preschool development which should be taken into account in practical situations of child management. The 2½-year-old child is very likely to be misunderstood. He has a reputation for going to contrary extremes. Parents frankly regard it as the most trying age in the whole preschool period. The spanking curve tends to come to a peak at about this time. There would be less spanking if parents understood the problems of development with which the 2½-year-old is confronted. He is outgrowing his infancy; in another half year he will be 3 years old. But in the interval, his whole behavior equipment and personality make-up are undergoing complex reorganization. He has not yet learned

to make a distinction between opposites; every pathway and culture, therefore, is a two-way street to him. Life is charged with double alternatives. His action system likewise is a two-way system. He is both so inexperienced and so immature that he does not make a clear-cut differentiation between *yes* and *no, come* and *go, give* and *take, grasp* and *release, push* and *pull.* His interest in these double alternatives is so evenly balanced that he goes from one extreme to another. He is confused, confusing, and contradictory. He is not sure of himself. What he wills to do, he cannot; and what he can do, he will not.

At present, his capacity for voluntary choice is weak; so he chooses both alternatives, not because of downright stubbornness, but because he lacks facility in balancing alternatives and of thinking of one alternative to the exclusion of another. Instead of following one line, he follows two (and one of them seems obstinate to the observer, who, by the way, rather pertinaciously, insists on only one. [The adult is pertinacious, not stubborn!]). The nerve cell organization which presides over inhibition is poorly developed. This shows itself even in such "neurological" actions as grasping and releasing. He does not have his flexor and extensor muscles in check and counter check. He tends to grasp too strongly, and he releases with overextension. He has not learned to let go. He has difficulty in releasing readily to go to sleep. And when he sleeps, he may even show a tendency to sleep too much. Similarly, he may not easily release the sphincters of bladder control, and so he withholds elimination too long—another instance of going to extremes which can scarcely be set down to innate wilfulness.

The peculiar limitations of his action system, therefore, account for his characteristic inability to *modulate* his behavior. He has such difficulty in making transitions that he tends to dawdle, as though it were hard for him to go from the familiar to something different. He is so conservative that he combats innovations. He wants to have things done the accustomed way. Sometimes he is actually a ritualist, particularly at home, and insists on having things *just so.* Since he is not sure of himself, he thinks that his salvation lies in his routines. These are his old and established self, the routines he has mastered. Accordingly, he insists on them with spirit and repetitiveness. He converts them into life-preserver rituals. If these rituals are wisely handled, they will in time lose their vitality. The child should not be punished on account of these rituals; their significance should be seen in perspective. Only a reasonable amount of patience is required, because in a few months this child will be 3 years of age, and typically he becomes almost a model of self-containment.

At the age of 3 years he has attained a power of choice through winning his battles with warring opposites. Accordingly, he is now more sure of himself. For a brief period, his whole action system is in relatively good working equilibrium.

To foresee the behavior characteristics of the 2½-year-old in terms of 3-year-oldness serves to make child management more intelligent and more humane. Without the developmental perspective, it becomes a temptation to use injudicious disciplinary measures. Parents insist that the 2½-year-old child understands what you say, and they insist that if he does not, he should understand. The temptation is to punish him if he shows resistance to the parent's perfectly reasonable demand which is made of him. If we understood the inner psychology of the 2½-year-old, we should appreciate that his resistance is neither obstinacy nor downright perversity. Development does not proceed in a straight line, nor is the organism in uniform, stable equilibrium. Developmentally, the organism periodically passes through stages of temporary disequilibrium when opposing forces are somewhat evenly balanced. When a developmental balance is attained, the organism reaches a stage of relative equilibrium. It then passes on to another stage of transient disequilibrium. This again is followed by a more stable stage of recurrent equi-

librium. Therefore, the so-called "perversities" of the 2½-year-old take on meaning when seen in the perspective of maturity.

The pediatrician can assist the parent to see the problems of psychologic care in such perspective, the perspective affording both comfort and insight if it is carried forward with the assurance that the 3-year-old will have himself in hand. A developmental philosophy humanizes the methods of child management and promotes and tends to protect the mental health not only of the child, but also of the parent. It reduces tensions; it increases a sense of security; it displaces the fidgety and meddlesome discipline; it facilitates guidance. The task of the physician is to impart and encourage this philosophy by giving concrete instructions concerning the nature and characteristics of mental growth. Otherwise the term *growth* is an empty abstraction.

The Mental Health of Adolescent Children

Clifford D. Sweet, M.D., Oakland, Calif.

Lately we have heard much of the adolescent child and his problems of adjustment to the world in which he finds himself. We have also had much emphasis upon the part the pediatrician should play in the guidance of parents and adolescent children toward a more perfect understanding of each other.

The adolescent child is important. There are 14,000,000 in the United States, only one-half of whom are in school, with 200,000 who are illiterate, 300,000 who are listed in the census as feeble-minded, and 15,000 who are in insane asylums. About 4,500,000, many under 16 years of age, are employed.

In passing, I record that I have seldom, if ever, been consulted about a serious behavior problem or a family conflict that concerned a regularly employed adolescent child; and many times when I have made later inquiry concerning one about whom his parents were anxious while he was in school, I have been answered with, "Oh, he's at work now, and he is all right." From this observation several conclusions such as the following might be mentioned: (1) the release from autocratic, too detailed parental authority follows naturally a degree of financial independence; (2) the self-respect grows at once in one who becomes a contributing member of any group; (3) the parents have in some instances finally given up their vain attempts to make "a silk purse" and have learned to accept with reasonable grace a good normal "sow's ear." I was consulted about a boy who made good grades only in printing because he was determined to become a baker while his father insisted that he must graduate from college since bakers stand no higher in the social order than did he, a plumber.

The pediatrician has an important part to play in all that concerns the adolescent child and his family, but however important his place in this field may be, it must not be entered at the cost of weakening his endeavors for the entire welfare of babies and preadolescent children. The obvious reasons upon which this statement rests are as follows: (1) No one can readily and fully understand adolescent children unless he has "grown up" with great numbers of them from babyhood until he is at least an adolescent in the practice of pediatrics. I have often wished that all high school teachers, or at least those serving as advisers and disciplinarians, had of necessity taught all the earlier grades. (2) The adolescent period is late for beginning the fundamental training of a child in the technique of living. An adolescent who is still using the crybaby method in order to gain his ends and who has gained little in understanding or controlling his emotions may be difficult to bring up to his chronological age. However, one must never give him up but must use all one's skill and courage to win him and his parents to an about-face that can yield brilliant results.

On the other hand, any normal child who has intelligent parents to whom his growth and development have been constantly explained by wise, sensible pediatricians

will pass through adolescence in stride to emerge a loyal, affectionate member of the family, well-advanced in the development of a personality that will serve him and society so long as life may last. The three periods into which adolescence has been divided—the early or ferment period, the mid- or crisis period, and the last or reconstructive period—will not stand out distinctly even though the endocrines are fermenting and the emotions, though powerful and at times difficult to understand, will produce nothing that approaches a crisis; and so little reconstructing will need to be done that priorities and subsidies will not be needed.

How, then, in simple language can we as pediatricians state to parents the fundamentals upon which a satisfactory family life must be built? And with few exceptions, strong, beautiful, fruitful, and therefore happy personalities arise only from a childhood that has been enriched by the example set by parents and fed by early memories in homes in which love, generosity, and honesty of statement and purpose have been the rule and ideal.

First, the child must be explained to his parents; and next, the parents must be taught to meet intelligently the changes that come within every child as he follows the usual pattern of growth and development.

Every intelligent child at birth begins to try to satisfy his needs and desires, and this effort continues as long as life and intelligence last. Early in life his needs and desires are simple and few—food, sleep, and rest, a temperature suited to his individual comfort, freedom from discomfort externally and internally, and an increasing amount of affectionate human companionship (well called "mothering" but which should also include "fathering"). In keeping with these simple needs, he has only crying to call attention to these needs. Crying, which is entirely normal for the infant, becomes "bad" only when, like the nursing bottle, its use is continued long after speech and self-help should have largely displaced it.

Since all human beings strive to get satisfaction for their needs and wants, the only differences among any number of them lies solely in the methods each uses. Here, then, is the great opportunity for parents to help their child develop a reasonable personality by steadfastly refusing to let disagreeable, unfair behavior become the most effective means of attaining desired ends.

Since no young child has had experience, he must constantly experiment with everything in his environment and be guided, as is every man of science, by the results obtained. He has no preconceived ideas so can be depended upon to discard that which is useless. A spoiled individual of any age is one who uses disagreeable methods in order to get what he wants too much of the time. Also, no child has regard in the beginning for anyone except himself and so is persistent and contents himself with a percentage of contests won, which is never satisfactory to an older person. This, combined with his entire lack of any rules of fair play, makes him a formidable opponent for any adult who does not clearly understand that he can hit below the belt, kick, bite, or scratch without any compunction.

The most common fault I find in parents is that, because they have little understanding of their child, they do not grow sufficiently in wisdom and understanding but remain almost static in their ideas and methods while the child is developing rapidly. Evidently, methods that are satisfactory for the infant will not be adequate for the child who is only 1 or 2 years of age and even less so as he reaches the age of adolescence.

Parents must learn to release their growing child gradually but steadily from the minutiae of their authority and at the same time keep a sensibly tight rein in all matters in which the child's experience is not yet an adequate basis for sound judgment. I heartily approve the motto of a camp which my younger daughter attended: "The girls in this camp may do anything they wish, provided it is not dangerous or unfair to their companions."

Also, as parental authority diminishes, an ever-increasing responsibility must be placed upon the child, not only for his conduct, but for the *esprit de corps* of the family and for its entire place, reputation, and standing in the community. I have never known a normal human being of any age who was not lifted above self, generally by the fact—not the hollow pretense—that he was a contributing member of his family, team, society, or gang, regardless of its name or title. In an earlier paper and in daily teaching I have compared satisfactory human relationship to a seesaw which works only when the length of the fulcrum and the weight of the person on either end make a like product.

Many times I have helped parents by asking them: "Have you ever known another adult with whom you spent any considerable length of time, however happily, who did not frequently irritate or displease you? Can you constantly please any other person, or for that matter, are you always at peace with yourself?" The answer is invariably "No," and I then ask: "Can we in fairness ask a child, as we too commonly do, to please us constantly? Must we not be at least as ready to appreciate the joy he gives us and overlook his faults as quickly as we do for our adult friends?" His faults are so often temporary while like behavior in an adult is chronic. Also, many of them are not faults but only his individual way which differs from ours and may, therefore, be better. "You may give them your love but not your thoughts, for they have their own thoughts."

1. I was consulted by a family because a boy of sixteen was going out every night. He was a fine healthy boy, well up in his classes, and a star on the high school track and basketball teams. Every evening at dinner he announced that he was going out, and his parents chorused, "You are not." I advised the withdrawal of the parents with the best face possible, and one month later, the report was, "He goes out only one or two nights weekly."

2. A boy and his father were in conflict because he did not get home from Saturday night parties at the midnight deadline set by his father. The father readily admitted the boy's excellent conduct as a senior in high school and his efficient help on afternoons and Saturdays in the family bake shop. However, he had to say "No" to one question I asked him, "Would you, when you were your son's age, have told your best girl that you had to go home before the last dance because your father said you must?" and his case was nonsuited at once.

3. A front door key for a well-grown boy as a symbol of his well-earned progress toward manhood instead of wakeful, reproachful parents made another family happier than they had been for some time.

DISCUSSION

DR. L. G. PRAY, FARGO, N. D.—What is the effect of restraint during sleep on the personality of the young child? I refer to the effect of sleeping bags, harnesses, and the like, which are used to keep the child lying down.

DR. SWEET.—I am opposed to harnessing children in bed. I have seen fourteen dead babies as a result of various types of restraints. If the child is not kept too warm with excess covers, he will not need restraints. For children who get uncovered I advise a garment made out of a discarded oversize bathrobe with long sleeves, the bottom of the garment sewed up and fastened in front with simple snaps. This assures warmth without restraint. I advise putting the child to bed for a rest period, not a nap. For the child who climbs out of bed, a fish net fastened over the crib works very well.

DR. PRAY.—Is not the sleep of the child disturbed by getting wet at night?

DR. SWEET.—The use of "soakers" works very well in keeping him more comfortable at night. The soakers absorb most of the excess moisture. If the

hands are kept warm, the child rests better. Another reason for discomfort is the presence of the ammonia diaper, resulting from using diapers that have not been boiled and rinsed. Most children wake several times during the night, which is normal. Wakefulness at night may also become a habit.

CHAIRMAN CASPARIS.—If a child gets out from under the covers, there is some reason for it. Usually he is too hot. Children need much less covers than the adult. Getting cold in bed is not so common as getting too hot.

DR. F. R. JANNEY, WAUWATOSA, WIS.—There seems to be an increasing number of parents who complain of difficulty in getting the adolescent child home early at night. How do you handle this problem?

DR. SWEET.—Mrs. Sweet and I have raised three children. Our rule has always been that on school nights the adolescent does not go out. Over the week end he may go out at night but there must be set rules for getting the child home. Of course we must expect to encounter protests such as, "I won't do it." This is normal and no attention is paid to it. We hold firm to our decision and the child conforms. We always try to make sure where the child is going at night.

CHAIRMAN CASPARIS.—This brings up the question regarding rules and flexibility in rules. Children are appreciative in adapting themselves to family rules and regulations. "Treat others as I would like to be treated" is a good rule to follow in handling adolescent problems. Somebody must have been tolerant with us in helping us to get through this difficult period. There has to be law and authority. Adults as well as children like authority.

DR. SWEET.—The teacher my child liked best was the one she described by saying "she's tough but she's fair." Being a parent is the hardest job I know. I have to go to others for consultation and help regarding my own children. The father must consult frequently with the mother regarding their children. Both parents must cooperate. I often ask my wife, "Why did you make that decision?" It is very important that the father come back into the family if we are to get the best results.

DR. M. C. CARLISLE, WACO, TEXAS.—In a family where there are two types of children what do you do to "bring out" the timid one?

CHAIRMAN CASPARIS.—One child helps to bring out the other child in the same family. The timid child has to be encouraged. He must get experience and confidence in doing things. When I get a group of new interns who might be called baby doctors, one intern has to be "sat on" to get the best out of him, while the timid type has to be encouraged.

DR. SWEET.—Someone has said, "The brave man is the coward who didn't run away." Fear is a normal emotional response.

DR. WELFORD.—During the present war crisis what procedure will help to safeguard mental health of children? I recently saw a child who wakened every night screaming because he was sure there were Jap bombers over his house.

CHAIRMAN CASPARIS.—War is merely a trigger that acts to bring out the background and environmental problems that were already impending. In such a situation there is already a background of trouble. The family gets alarmed and have already created an atmosphere of fear which reacts on the child, who in turn becomes fearful. One thing we have felt is that children are more important than war work. If as much time were given to raising children as is spent in war effort, there would be less trouble with children.

DR. SWEET.—One word about fears. Fear in children is not kept alive unless by parents. Parents must instill confidence in the child. War is a challenge. If we prepare a child before tonsillectomy by telling him exactly what is going to happen and that he is going to have pain, he will be much less fearful.

CHAIRMAN CASPARIS.—Even adults want sympathy and understanding in situations such as operations. There are two different make-ups in human beings. First, the cautious or sensitive type; second, the "go out and do anything" type. Similarly there are those in the same family who have anxiety regarding the war and those who have no anxiety.

CHAIRMAN BEVERLY.—Medical students of today have a reasonable amount of training in pediatric psychiatry, but they are still unable to coordinate these subjects and are therefore unable to tell how to train children so as to preserve mental health and physical health.

Security to the child is given by the parents who want the baby. The parents, the mother, the father, the home, the country, the school, and the church all help him to develop patterns so that he can meet life as it is and adapt himself. The important facts will now be discussed. Dr. Montgomery will discuss the mental and physical factors, and Dr. Aldrich will tell us that the mental and physical factors are not separate.

DR. MONTGOMERY.—Dr. Beverly has earmarked the discussion. The physical and mental do fit together very intimately. The rate of development of the physical and mental aspects are important. Let us consider, first, the parents' physical, mental, and emotional development, their aptitudes, hostilities, and how these react upon the child, the influence of home, school, the nurses, etc.

The modern child must assume in the community the changes in our culture. It is no longer culturally necessary for a mother to nurse her child. She has no milk. The hospital imposes two facts: (1) material, involving warmth, food, and shelter; and personal or interpersonal. These may become very complex; one must feel that his part in society is secure for him. The interpersonal level involves active participation. He must feel that he is deservedly wanted. Affection and love involve both giving and receiving.

Breast feeding gives an outlet for this definite sharing of security. Each needs the other and each is essential. There is the instinct to establish personal relations. Love implies something separate from affection. A breast feeding schedule must be adopted to the infant's rhythm.

There is an educational element in breast feeding—the effect upon the mother in continuously giving to the child; the mother feels the compulsion to nurse her child. This is not a neurotic magnified need. Hostile feelings are ameliorated by nursing the baby from the breast.

CHAIRMAN BEVERLY.—The baby needs its mother and the mother needs her baby.

DR. G. R. FISHER, COLORADO SPRINGS.—Should a baby nurse every hour or every six hours?

DR. MONTGOMERY.—During the newborn period a reasonable frequency is four hours but not exactly every four hours.

CHAIRMAN BEVERLY.—Should he nurse twelve times a day, change schedule every day? Two hours? Four hours? Or six hours?

DR. FISHER.—Should not the mother regulate the routine?

DR. MONTGOMERY.—Mother and babe should work out their routine together.

CAPT. G. W. ESTY, U. S. ARMY, M. C.—In anticipating her schedule the mother does not know the difference in the cry, whether it is from gas or hunger. Is it her privilege to offer the bottle every time he cries? The baby may take the bottle, 1 ounce, hourly. The mother develops a nervous breakdown. The mother must instinctively be able to tell the difference. It is the responsibility of the baby to tell how much he needs.

CHAIRMAN BEVERLY.—Dr. Aldrich would have the baby in the mother's room and then she could tell all these needs.

CAPT. ESTY.—If baby does not fill up, he may waken each hour. One of my patients nursed 7 ounces when in the hospital and refused to nurse at home.

CHAIRMAN BEVERLY.—How can you train the mother to know?

DR. ALDRICH.—Tell the mother that the baby cries from heat, cold, and hunger and will awaken for his social period, bowel movements, and urination; the mother must learn to recognize his needs.

Dr. Beverly has just defined security as the feeling the child gets when he is born of parents who want him and are willing to allow him to grow up according to his own rhythms. If we accept this as being an important objective in rearing children, it may be worth while to consider for a few moments its second aspect—growing up according to inherent rhythms.

As we understand the situation of the newborn baby now, in our early stages of study of growth and development, a young infant exists largely through his inborn capacity to control his essential functions on a reflex or subcortical basis. In this way he adjusts the speed and rhythms of such vital functions as breathing, circulation, digestion, eating, sleeping, and elimination.

The first three of these are so obviously under complete automatic supervision that no one has had the temerity to tamper with them. But the latter, eating, sleeping, and elimination, are more easily subject to external control which has been inflicted upon him, sometimes in an almost ruthless manner, much to the detriment of the organism. I would like to suggest for purposes of discussion here that it is just as important that we provide a favorable environment for the growth of eating, sleeping, and elimination as it is to provide pure air and proper food elements for the best operation of breathing, circulation, and digestion.

Eating, sleeping, and eliminating are all rhythmic in their action under the influence of internal controls. When these forces act naturally, each performance results in a pleasurable sensation which gives, in a sense, a feeling of satisfaction and competence or security. As a baby grows, the innumerable repetitions of such successful acts produce a feeling of inherent ability to function successfully in the world. Unless a person fels competent to manage such intimate details as what and when to eat, how long and when to sleep, and when and where to eliminate, I doubt if he can claim much security.

It would be a hopelessly discouraging list if I attempted to mention all the ways these functions are tampered with, but just by way of setting up something to "shoot at," the following might be mentioned: regimentation of eating and sleeping times, restriction as to the amount and kinds of foods, early artificial feedings instead of breast milk, the unnecessary awakening of babies for routine procedures, and premature training in bowel and bladder habits. Such interferences occur scores of times daily in the lives of most modern babies in civilized countries. They are responsible for much of the confusion and insecurity of human adults today.

Before we open this subject for general discussion, it might be suggested that it is this same tampering process which is responsible for most of our "spoiled brats." Typical spoiling takes place when a child learns a nasty way to get what

he inherently needs after his legitimate demands have been ignored. Parental indulgence enters the scene after, not before, the spoiling occurs.

DR. M. M. MILLER, AKRON, OHIO.—On the other hand, do you not think it possible to handle babies too much? It seems to me that the very young infant's primary requirements are for satisfaction of his hunger and for sleep, and he should be encouraged during this period of rapid growth to get as much sleep as possible. For that reason I tell my mothers that the only times they should handle the very young baby are when they bathe him, change his diaper, pick him up to feed him, and when they burp him. Make sure his hunger is satisfied, that he is warm, dry, and comfortable, and then let him alone. I do not mean that babies should not be loved, cuddled, and fondled, but that this can be overdone to the detriment of the child. I see lots of babies whose sleep is disturbed and who are restless, irritable, and spoiled to death by too much handling.

CHAIRMAN BEVERLY.—Mother should wait five minutes to see if he moves his bowels; if he does not, then feed him. You may have to experiment a bit on the child. For example, a baby was on a two-hour schedule and the family was a wreck, but when he was put on a four-hour schedule all went well.

DR. ALDRICH.—The most irregular baby I ever saw and the "cryingest," I managed this way. I asked the mother to nurse the baby whenever he cried, but not to tell the neighbors. I did not hear from her; I called her two weeks later and found that everything was going smoothly. This same mother called when the baby was 3½ months old. She said, "If we take the baby into the living room after supper while we read the paper, he does not say a word, goes right to sleep, and sleeps all night, but if we put him in bed right after supper in his own room, he will cry all night, and I mean cry all night."

DR. GESELL.—What is the theory behind the self-regulation schedules?

With us, it is not altogether a matter of theory. As reported elsewhere,* we have shown that when a baby is given an adequate chance, he works out a schedule appropriate to his peculiar needs and rhythms. Every living organism is a storer and distributor of energy. The human infant is no exception. His daily cycle of activity and sleep reflects the way in which he husbands and expends his energies. These energies are primarily determined by physiologic factors, and only secondarily by cultural factors. The self-demands and cultural demands must somehow be brought into mutual accordance.

The newborn baby is, in many respects, physiologically awkward, but he is also physiologically wise. He shows his wisdom in self-regulatory fluctuations. Development does not proceed in a straight line. The fluctuations are not undesirable irregularities. They are effortful attempts on the part of the organism to accomplish increasingly mature adjustments. For these reasons, we should give the infant a lead in initiating variations from day to day, because through these variations, he tests his powers and achieves his adjustments. We assist him by setting up a provisional schedule. But we cannot arrive at an optimal schedule without granting him latitude for fluctuations.

During the first quarter of the first year, the fluctuations of the child's feeding and sleeping demands are most numerous and conspicuous. The observant mother learns to interpret these demands judiciously from day to day. She takes her initial cue from the time of the morning waking; she recognizes hunger fretting; she permits him to sleep as long as he wishes and allows him to wake of his own accord. She is willing to shorten or lengthen the intervals between feedings in terms of the

*Gesell, A., and Ilg, F.: Feeding Behavior of Infants: A Pediatric Approach to the Mental Hygiene of Early Life, Philadelphia, 1937, J. B. Lippincott Co., XII, 210 pages.

child's sleep and hunger demands. By individualization of feeding, the infant is most directly and completely satisfied. He is satisfied vegetatively and emotionally. He escapes periods of want, anxiety, and distress. By satisfying his organic needs, we nourish not only his body, but also the sense of security which is essential to mental health.

DR. MILLER.—In my experience I have not seen any emotional damage or psychologic maladjustment result from not feeding babies at the breast. On the contrary, it seems to me that breast-fed babies are in many cases more spoiled, maladjusted, and harder to manage when the time comes to put the baby on a mixed diet than is the case with properly regulated artificial feeding. I feel that breast feeding is more important to the mother's emotional pattern than it is to that of the infant, for the mother who nurses her baby is apt to form a deeper emotional attachment to her baby than the mother who feeds her baby with a bottle. It seems to me the young infant's emotional need for physical contact has been overstressed. His primary need is satisfaction of his hunger pains, and this can be satisfied just as well, and, in many cases, better by proper artificial feeding.

In any case, whether breast or artificially fed, the schedule should be adjusted to the baby's natural rhythm of sleep and hunger; that is, instead of a hard and fast three-hour or four-hour schedule, it would seem better to employ a flexible schedule of, for example, every three to four hours, according to the infant's appetite and sleeping habits.

DR. GESELL.—Does not the self-regulation plan make the child too irregular?

Not in our experience. There are individual differences, and it is these very differences which we wish to recognize. Some babies on a self-demand basis settle down very early to a rather smooth and regular schedule. Others fluctuate a great deal, but these are the very babies who may benefit most from self-regulatory schedules. Their greater tendency to variation from day to day may represent constitutional characteristics and a natural mode of development. If the self-regulation policy is wisely administered, such fluctuations are not caused by the environment but express the natural needs of the child. Feeding difficulties and other difficulties may be induced by overrigid insistence on a regular clock schedule. Hygienic psychologic care requires consideration of the self-regulatory fluctuations.

How does the baby register his self-demands?

By his behavior. He expresses his well-being and his ill-being in his modes of behavior. He does not have words at his command, but he has two sets of signs and signals, the negative and the positive—those which express avoidance and rejection and those which express seeking and acceptance.

The negative signals include crying, fretting, uneasiness, refusal, and anxiety. The positive signals include quiescence, relaxation, satiety, cooing, smiling, and pleasurable self-activity. An inflexible schedule based on a more or less arbitrary time interval would ignore the infant's signs and signals. It would insist on regularity of intervals in spite of the infant's irregular fluctuations. It would require the mother to wake her infant when he would insist on sleeping.

When should self-regulation begin?

Ideally, the policy of self-regulation should begin with the birth of the baby, whether he is breast fed or bottle fed. Breast feeding naturally has some advantages.

In the lying-in hospital, self-regulation can be accomplished through a simple and flexible rooming-in arrangement. Under this arrangement, the baby may be brought into the mother's room and put in a secluded corner, or, more intimately, at the foot or side of the bed in accordance with the mother's strength and wishes. After the evening or night feeding, the baby might well remain in the nursery. The presence of the baby through the daytime gives the mother an increased sense of security,

and it puts her in a more favorable position to build up a sense of relationship with the infant and a sense of familiarity with his needs. She, of course, has the help of the nurse, but the incidental oversight of the mother makes her perceptive of the self-demands of the infant, all of which increases the advantages of a self-regulatory program of care on which she and the physician have embarked. The arrangement must, of course, be kept under the supervision of the physician. The rooming-in self-regulatory arrangement is not rigid. It is flexible and can be altered to meet the needs of both mother and child from day to day. It has other incidental advantages which come to realization when the mother leaves the hospital and takes over at home.

DR. ALDRICH.—Babies are not spoiled by agreeing with them but by being tough with them. If a baby learns, instead of being spoiled by loving, to fill his needs the nasty way, for learn he must some way, then you have a problem.

DR. BOYD.—Are not many of the problems the mother's problems?

DR. ALDRICH.—Yes, but we can teach her that the baby will develop acceptable food habits in from six to twelve months. Of course all babies differ; for instance, if after two minutes' nursing you turn his head away or extend his legs and hold them, one will scream and resume nursing, another will refuse to resume nursing, another will smile and play, another will just lie there. They just are different.

DR. GESELL.—We have been able to see what institutions do to some children; some are robust enough to take it, but, on the whole, institutional care is an insult to the child, and the child will show it in eight weeks' time. By 28 weeks of age he becomes timid, withdrawing, blandfaced; by 40 weeks he is retarded in talking. You can see the same result in the oversanitary or overmodern home.

CHAIRMAN BEVERLY.—Do you want to rock babies?

DR. ALDRICH.—No, but I like the mother to rock him if she wishes or if the baby wants it.

CHAIRMAN BEVERLY.—I got the impression you wanted to.

DR. ALDRICH.—I did when I was young.

Let us work it out with the mother. She may have to keep it up for a while, but the child will not care to be rocked at 14. No, I do not think baby wants to be bounced on your knee.

QUESTION.—Would you let a baby cry for three nights? It seems to work and baby seems happy.

DR. ALDRICH.—It all depends on how much affection the baby is getting, you cannot teach them without affection but you can show them occasionally that they are not socially acceptable. It is dangerous, though, for too often their need is not met.

CHAIRMAN BEVERLY.—Yes, they must be trained, but not by 6 months; more acquire a sense of reality as an individual by 1 year. What patterns should be taught; that is, regarding a baby who cries at 1 A.M. or 6 or 10 P.M.? That is common and he may cry it out in three nights but it also means, if the child needs affection that he should get it during the day. Take him up at a reasonable time before feeding and fondle him; let him play stripped. He is more apt to be quiet after 6 P.M. It is also a good thing to allow the baby to go ahead on his own steam, to get a physical workout.

DR. ALDRICH.—The whole setup of bassinet and frills is very abnormal. Children are not normal at this age, although they can take it, if fundamentals are right.

CAPT. ESTY.—Babies should be given four responsibilities when they are born. Interference with these natural responsibilities will ultimately result in the majority of our pediatric problems.

First, there is the responsibility of the baby in deciding how much or how little he wishes to eat at any one meal. If a baby is properly handled, and the technique of feeding is well understood by the mother, a baby will drink close to what he really wants and needs and will not overeat in most cases. If the mother believes that because the doctor ordered 4 ounces in each bottle the baby must have this quantity, she at once develops an attitude of emotionalism, stress, strain, and worry concerning food, which will ultimately lead to the child who "won't eat."

This viewpoint is now fairly well understood and followed. Dr. Aldrich's idea of allowing the baby to decide when he wishes to eat may be right theoretically, but it is fraught with considerable difficulty for the mother to carry out intelligently. It takes an expert to tell whether a baby is crying for food, pain, attention, discomfort, or boredom. If the mother is given the privilege of feeding the baby when he acts hungry and cries, she will repeatedly offer the infant the nipple in order to keep him from crying, with the result that she will soon use the bottle as a pacifier, and the baby will soon look for it in that role. I have seen babies whose mothers were feeding them an ounce every hour instead of perhaps 4 ounces every four hours. This continual fussing with the child results in interference with sleep habits and leads to nervousness from overstimulation. In other words, the overhandling of the baby in its early weeks not only results in a demanding and irritable baby, but also an exhausted, discouraged mother, an experience which may color her attitude toward her child for years to come. I do not advocate a rigid stop-watch type of schedule. Flexibility is essential. The baby may be fed at eleven o'clock at night instead of ten, if desired, and he should never be awakened at 2 A.M.; otherwise, he will accustom himself to receiving food at that inconvenient hour. Any time up to 4:30 A.M. would be considered a 2 A.M. feeding. From 4:30 on would be a 6 A.M. feeding advanced. In this way the baby eliminates his own 2 A.M. feeding when he is ready. However, I do advise mothers to anchor their schedule at about 2 P.M. and at 6 P.M. so that the cycle may not be entirely changed from day to day, causing difficulties in the mother's own routine.

Allowing babies to decide when they wish to eat, regardless of schedule, is very much like the laissez-faire type of education, where the teacher interferes as little as possible with the child's behavior, letting him do what he wishes when he wishes. Children need guidance and a firm kindly hand to lead them and to give them security. And so it is with the infant. The baby without any guidance fails to learn, continues to act in a rudimentary manner. If one argues, as Dr. Aldrich has done, that the young infant should have to experience no frustrations or unpleasantness and that his every desire should be gratified, just when does learning through experience begin? The word discipline means learning. Learning is a positive thing. Discipline should also be a positive educational approach, and not merely, as so many parents believe, a negation. Discipline in the infant, then, is synonymous with learning through trial and error and experience. The first disciplines of infancy concern themselves with patterns of behavior adapted through experience in his relationships with his parents. The development of character and personality starts at birth and it is a give-and-take proposition. To be balanced, it cannot be all give or all take. Giving a baby security does not mean that his contacts with life must be free from every unpleasantness. The metaphysicians tried that experiment with their famous baby and failed. In other words, giving the infant the responsibility of deciding how much he wants to eat is an easy and practical thing

for the average parent to allow, but giving him food whenever he may "want" it is much more difficult, because it requires a knowledge on the part of the parent to interpret the baby's "want." For development to proceed, nature fortunately provides a flexible being so that adaptation can occur without injury resulting.

The second readily allowable responsibility for the infant is his determination of his bowel rhythm. Interference assisted by artificial means results often in stubborn constipation. The third responsibility is allowing the baby to urinate and defecate at will without the commonly overemphasized "early training." The fourth responsibility is that of sleep. You may give the infant the chance to sleep, but you cannot make him do so.

Regarding the questions of crying at night: "What to do about it?" "What is the cause?" Many babies are awakened by a bad dream which scares them and causes them to cry. To allow the baby to cry without investigation would be dangerous not only because some accident might have occurred, but also because at such a time the child needs security. If not given, the baby might soon associate falling asleep or awakening with something to be afraid of, resulting in a continuation of difficult behavior. The baby should be soothed and comforted as well as possible, but excessive emotionalism and concern should be avoided, because then the baby soon learns that a repetition of the act brings about a similar reaction on the part of the parent, and a new attention-seeking device is born. In short, a parent cannot give a feeling of security to a child and be insecure emotionally herself.

The causes for these bad dreams and other nervous discharges taking place during a child's sleep are usually due to overnervous fatigue and stimulation. This results often from allowing a too late retiring hour after the normal fatigue line has been passed, and perhaps a bath before supper rather than before bed in order to save time should be tried. The father must be warned not to excite and roughhouse with the child at these times.

DR. GESELL.—I have a feeling that there is a good deal of underlying agreement and in the right direction. You cannot undo the principle of individualizing the babies—love, sleep, food, etc. Babies are very wise; the wisdom of the body and the mind allow fluctuations like a man walking a tight rope—first this side, then that.

DR. ALDRICH.—When babies' needs are not met, it is up to the doctor to find out why not; you must promptly satisfy needs, for baby has not much capacity for delayed reaction, but gradually he will acquire some.

DR. J. L. KOHN, NEW YORK.—An infant, about 14 months old, begins to awaken frequently during the night. As far as one can ascertain (a) there is no physical cause for the crying and (b) there has been no change in the immediate environment. Let us assume that there has been no arrival of a new baby and that the child is accustomed to the surroundings of his own room. How should this situation be handled? Should (1) the parent go to the child's bed, soothe, and talk to him every time he awakens or (2) should the parent use mild disciplinary methods?

DR. ALDRICH.—I would feed him; I would try to find what the child wanted and try to supply it.

DR. GESELL.—Is it normal for a baby to cry constantly? Constant crying is often one of the symptoms of amentia.

DR. KOHN.—Assume now that the methods suggested have not been effective. Is one justified in giving the infant a milk sedative, such as ½ or 1 gr. of phenobarbital for several nights? In this way both child and parents get a welcome night's rest.

CHAIRMAN BEVERLY.—He is obviously frightened, has bad dreams. Take him to mother's bed and get him over it. It may take three weeks, but go as far as necessary in giving the baby the security he needs.

DR. ALDRICH.—Colic—atropine—frequent feedings—that is a medical problem. Let the mother care for her baby and let the 45-year-old nurse do the housework or go to the factory. Should he sleep alone? Perhaps after a few weeks. Maybe the baby cries because it does disturb the mother.

DR. GESELL.—One factor begins at about 2 years of age—ritualism. A certain pattern has to be followed. He is learning so fast he needs that ritualism to get released into sleep.

DR. ALDRICH.—We had to struggle to get into the final draft of the White House Conference report the statement that "the basis of the whole social structure is making the baby secure."

Occasionally, the mother must keep the child dependent on her, but that is not normal affection. You just have to keep the premature infants in incubators, but you can give them breast milk.

DR. GESELL.—I would leave young mothers where they belong. Discourage employment of mothers of children under 5 years, especially under 2 years. The mother has the right to make the wrong decision—that's democracy—but we can have policies and counseling centers. They are important.

CHAIRMAN BEVERLY.—I have never seen trouble by indulgence, except when grandparents tried to rear the children themselves and thus frustrate the parents.

Parents often say, "Ever since my mother and father were here my baby has been a hellion." They can upset a baby in four days and undo all our efforts.

DR. ALDRICH.—There are two kinds of grandparents; those who think they did a good job on daughter and will not lack confidence in whatever she does, and the others who have always fought their children and will also fight their grandchildren.

The Pediatrician and the War

The following is a continuation list of Fellows of the American Academy of Pediatrics on active duty in the Military Services:

Army

Dr. Sam Phillips, Little Rock, Ark. (Major)
Dr. George Renner, Jr., Cincinnati, Ohio (Rank unknown)
Dr. David W. Goltman, Memphis, Tenn. (Captain)
Dr. Pascal F. Lucchesi, Philadelphia, Pa. (Major)

Navy

Dr. Abram Kanof, Brooklyn, N. Y. (Lieutenant Commander)
Dr. Charles I. Solomon, Meriden, Conn. (Lieutenant Commander)
Dr. John H. Doval, Sacramento, Calif. (Lieutenant, j.g.)
Dr. Matthew Lewison, Chicago, Ill. (Lieutenant)

Dr. Henry P. Wright, Montreal, Quebec, Canada, and Dr. E. S. Lippman, Minneapolis, Minn., have been discharged.

The following Fellows of the Academy have been promoted since entering the Military Services:

Army

Major Montgomery Blair, Jr., to Lieutenant Colonel
Captain John E. Brown, Jr., to Major
Captain Norman T. Crane to Major
Captain Herman W. Farber to Major
Captain Seymour Fisher to Major
Lieutenant Moe Goldstein to Captain
Lieutenant Benjamin M. Kagan to Captain
Major Lewis Webb Hill to Lieutenant Colonel
Major Samuel J. Hoffman to Lieutenant Colonel
Captain William A. Howard to Major
Major John P. Hubbard to Lieutenant Colonel
Major James Gilliam Hughes to Lieutenant Colonel
Lieutenant Howard M. Jacobs to Captain
Captain Robert E. Jennings to Major
Lieutenant Albert M. Jones to Captain
Major Samuel Karelitz to Lieutenant Colonel
Major George S. Littell to Lieutenant Colonel
Captain J. Keller Mack to Major
Captain Paul R. Meyer to Major
Major Philip A. Mulherin to Lieutenant Colonel
Lieutenant John J. Shields to Captain

Canadian Army

Major John R. Ross to Lieutenant Colonel

Royal Air Corps

Major Frederick F. Tisdall to Wing Commander

Navy

Lieutenant James E. Bowman to Lieutenant Commander
Lieutenant Sims A. Chapman to Lieutenant Commander
Lieutenant Commander David P. Evans to Commander
Lieutenant Commander George M. Lyon to Captain
Lieutenant Commander Joseph Palma to Commander

News and Notes

Dr. Frederic W. Schlutz, Professor and Chairman of the Pediatric Department of the University of Chicago, died March 8 of a cardiac condition. Dr. Schlutz has been particularly identified in recent years in developing pediatric relationships with our Central and South American colleagues. He had planned to leave March 10 for an eighteen-week good-will mission to Latin America for the State Department.

Word has been received of the death of Dr. Karlton G. Percy, of Boston, Mass.

The *Annual Meeting* of the *American Academy of Pediatrics* will be held in St. Louis, November 9-11, at Hotel Jefferson.

A research department of pediatrics has been established at the Children's Hospital of Buffalo with a grant from the Trustees of the estate of the late E. M. Statler. Dr. Edward M. Bridge, formerly of the Department of Pediatrics at Johns Hopkins, has been appointed to the Research Professorship of Pediatrics at the University of Buffalo and will have charge of the work at the Children's Hospital. The hospital is remodeling a floor of one of its buildings to house the new laboratory.

Dr. John D. Lyttle, formerly of the Department of Pediatrics at Columbia University, New York, has been appointed Professor of Pediatrics in the School of Medicine of the University of Southern California and has been appointed Director of Pediatrics at the Children's Hospital in Los Angeles.

Dr. Arild E. Hansen, formerly Professor of Pediatrics at the University of Minnesota, has been appointed Professor and Chairman of the Department of Pediatrics at the University of Texas School of Medicine at Galveston. He will also serve as Director of the University of Texas Child Health Program.

The following were certified by the AMERICAN BOARD OF PEDIATRICS at the examination in New York, N. Y., March 25 and 26, 1944:

Dr. Louis Thobro Campbell, 11 Kiwassa Rd., Aranac Lake, N. Y. (Now in Army)
Dr. Cornelia Morse Carithers, 1625 Riverside Ave., Jacksonville, Fla.
Dr. William Andrew Daniel, Jr., 115 S. Union St., Montgomery, Ala.
Dr. Thomas Albert Foster, 131 State Street, Portland, Me.
Dr. Mariana Gardner, 1850 Gilpin St., Denver, Colo.
Dr. A. Louis Girardin, Jr., 915 Menlo Rd., Fort Myers, Fla. (Now in Army)
Dr. Joseph Glassman, 444 Georgia Ave., Brooklyn, N. Y.

Dr. Henry Mitchell Glaubman, 20 Lenox St., Hartford, Conn.
Dr. Solomon Goodman, 8662 Bay Parkway, Brooklyn, N. Y.
Dr. John L. Green, 3501 Ryan Ave., Philadelphia, Pa.
Dr. Martin Green, 6022 Carpenter St., Philadelphia, Pa. (Now in Navy)
Dr. Joseph James Greenlaw, 1632 Rhode Island Ave., N. W., Washington, D. C.
Dr. Robert Dean Hart, 611 N. Sheridan Rd., Peoria, Ill. (Now in Army)
Dr. Akiba Joseph Himmelhoch, 19220 Kingston Dr., Detroit, Mich. (Now in Army)
Dr. Ng. William Hing, Puerto Rico (Now in Army)
Dr. Martin Joseph Hurst, 1102 Grand Ave., Kansas City, Mo. (Now in Army)
Dr. Gertrude Martina Hyde, 264 King St., Port Chester, N. Y.
Dr. Nina R. Lief, 1301 Cornaga Ave., Far Rockaway, N. Y.
Dr. Alla Dunewitz-Litvak, 1145 Eastern Parkway, Brooklyn, N. Y.
Dr. Lula O. Lubchenco, Children's Hospital, Denver, Colo.
Dr. Robert Alister McGuigan, 2419 Park Pl., Evanston, Ill. (Now in Navy)
Dr. Robert Dickie Nix, Mt. Lebanon, Pa. (Now in Army)
Dr. Israel J. Rosefsky, 8 Riverside Dr., Binghamton, N. Y.
Dr. Russell E. Sangston, 2 West Main St., Uniontown, Pa. (Now in Navy)
Dr. Sydney Milton Saul, 5736 Hobart St., Pittsburgh, Pa.
Dr. Lillian Shulsky, 165 Clinton St., Brooklyn, N. Y.
Dr. Theresa Ora Snaith, 450 Center Ave., Weston, W. Va.
Dr. Harold E. Stadler, 5819 N. Pennsylvania St., Indianapolis, Ind. (Now in Army)
Dr. Leon Sternfeld, 65 Court St., Buffalo, N. Y.
Dr. Alfred J. Vignec, 121 E. 61st St., New York 21, N. Y.
Dr. Richard Wagner, 370 Commonwealth Ave., Boston, Mass.
Dr. David H. Weintraub, 219 Bryant St., Buffalo, N. Y.
Dr. Arnold H. Williams, 7th and West Sts., Laurel, Del.

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